Aldrin and Dieldrin: A Review of Research on Their Production, Environmental Deposition and Fate, Bioaccumulation, Toxicology, and Epidemiology in the United States

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In the last decade four international agreements have focused on a group of chemical substances known as persistent organic pollutants (POPs). Global agreement on the reduction and eventual elimination of these substances by banning their production and trade is a long-term goal. Negotiations for these agreements have focused on the need to correlate data from scientists working on soil and water sampling and air pollution monitoring. Toxicologists and epidemiologists have focused on wildlife and human health effects and understanding patterns of disease requires better access to these data. In the last 20 years, substantial databases have been created and now are becoming available on the Internet. This review is a detailed examination of 2 of the 12 POPs, aldrin and dieldrin, and how scientific groups identify and measure their effects. It draws on research findings from a variety of environmental monitoring networks in the United States. An overview of the ecologic and health effects of aldrin and dieldrin provides examples of how to streamline some of the programs and improve access to mutually useful scientific data. The research groups are located in many government departments, universities, and private organizations. Identifying databases can provide an "information accelerator" useful to a larger audience and can help build better plant and animal research models across scientific fields. *Key words:* air, bioaccumulation, birds, breast cancer, breast milk, colon cancer, data sources, endocrine disrupter, fish, manufacturing, marine mammals, neurotoxicity, Parkinson's disease, pesticide, POP, reproductive, soil, transplacenta, water. — *Environ Health Perspect* 109(suppl 1):113–139 (2001). *http://ehpnet1.niehs.nih.gov/docs/2001/suppl-1/113-139jorgenson/abstract.html*

International Issues

The growing global volume of chemicals currently in use has raised concerns about their long-term effects on health and the environment. Between 1980 and 1995, annual use of global pesticides increased from 2.0 to 2.7 million kilograms. In the last decade, four international agreements have focused on reducing or eliminating production, trade, or use of a group of 12 chemical substances known as persistent organic pollutants (POPs).

Four International Agreements Focused on POPs

In 1994, 103 countries adopted the United Nations Environmental Program (UNEP) Washington Declaration on Protection of the Marine Environment and Land Based Activities Agreement, which calls for a reduction of 12 substances known as POPs (*1*).

This agreement paralleled work undertaken by the Third International North Sea Ministerial Agreement (*2*) to reduce emissions of priority hazardous substances by 50–70% from 1985 levels. In September 1992, the work to implement this agreement began. The Oslo and Paris Commission (OSPARCOM) asked Germany to oversee an atmospheric emissions inventory of POPs for the whole of Europe. The Netherlands Organisation for Applied Scientific Research (TNO) in the Netherlands completed this work for 38 countries in June 1997 (*3*).

In June 1998, the Convention of Long-Range Transboundary Air Pollutants (LRTAP) Agreement was ratified to control and reduce 16 POPs in 43 countries (*4*).

This meeting was followed by the Convention on Prior Informed Consent Procedures (PIC) for certain hazardous chemicals and pesticides in international trade, which was agreed to in Rotterdam, the Netherlands, in September 1998 (*5*).

Now the UNEP POPs negotiations are working toward a global agreement to ban and eliminate 12 POPs by the year 2001 (*6*) (Table 1).

Chemicals: global production and trade. The POPs group includes 12 substances, 9 agrochemicals (aldrin, chlordane, DDT, dieldrin, endrin, mirex, heptachlor (HCH), hexachlorobenzene (HCB), toxaphene), and 3 industrial substances (polychlorinated biphenyls [PCBs], dioxin, and furans) (*6*). This group is known as the "Dirty Dozen." Other substances have been added to the list of POPs of concern.

Production. The nine POP produced for use in agriculture were introduced from 1920 to 1950, with production peaking in the 1960s and 1970s for crops. By 1990, these substances were banned for use on crops and termite control by most North American and European countries, although they continued to be manufactured as a wood preservative, for pest control, and as intermediaries for other chemical processes. Production volumes of POPs in North America and Europe have substantially declined since the 1970s. Japan, not known to have been a major producer, produced smaller quantities of endrin, heptachlor, and HCB.

However, POP production and world trade still persist. There are about 39 countries where companies report production. There are probably three more countries—Tanzania, Lebanon, and Sri Lanka—where companies or foreign subsidiaries of producing countries, formulate POPs, but these countries have poor right-to-know public reporting requirements (*7*).

A rapid expansion of overall chemical manufacturing is occurring now in Asian countries. For the 30 largest chemical manufacturers, 14% of their growth has been in developing countries (*8*). Between 1980 and 1997, chemical production capabilities in developing countries increased by more than 300%. Many more locally owned chemical companies are reporting production for export and most developing countries with a chemical manufacturing base are now able to produce alternatives to POPs.

Today it is difficult to identify the volume of POPs produced by the companies listed in the trade by international chemical directories. In 1990, Battelle Corporation in Geneva, Switzerland, developed a database on POP production that became the basis for studies conducted by Environment Canada, the OSPARCOM inventory, and the U.S. Environmental Protection Agency (U.S. EPA)

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I acknowledge help of the World Wildlife in allowing me to use their collection of papers on the toxicologic effects of persistent organic pollutants, of G. Blodgett at the National Press Club Library, and of the fine staffs from the United States Geological Survey, National Oceanic and Atmospheric Administration, and U.S. Environmental Protection Agency for producing data used in this review article.

Received 25 April 2000; accepted 30 November 2000.

Table 1. International negotiations to regulate POPs and a list of substances under consideration for regulation.

UNEP POPs 12 substances, reduce/eliminate (103 countries)	LRTAP 16 substances, reduce/eliminate (43 countries)	PICs 27 substances, trade notification (95 countries)	OSPARCOM 27 substances, 50–70% reduction (38 countries)
Aldrin	Aldrin	Aldrin	Aldrin
Chlordane	Chlordane	Chlordane	Chlordane
	Chlordecone (t)	Chlordecone (t)	
		Captafol	
		Chlorobenzilate	
DDT	DDT	DDT	DDT
Dieldrin	Dieldrin	Dieldrin	Dieldrin
		Dinoseb: dinoseb salts	
		EDB	
		Fluoroacetamide	
Endrin	Endrin	Endrin	Endrin
HCH	HCH	HCH	HCH
Hexachlorobenzene	Hexachlorobenzene	Hexachlorobenzene	Hexachlorobenzene
	Hexachlorocyclohexane	Hexachlorocyclohexane (HCB)	
	Lindane	Lindane (included in HCH)	Lindane
Mirex	Mirex	Mirex	Mirex
Toxaphene	Toxaphene	Toxaphene	Toxaphene
		2,4,5-Trichlorophenoxyacetic acid	
		Phosphate	
		Tris(2,3-dibromopropyl) phosphate	
		Methylparathion	
		Methamidophos	
		Monocrotophos	
			Nitrofen
			Endosulfan
			Fenthione
			Pentachlorophenol
			Quintozene
	Hexabromobiphenyl		
PCBs	PCBs	PCBs	PCBs
		Polybrominated biphenyls	
		Polychlorinated terphenyls	
Dioxins	Dioxins	Dioxins	Dioxins
Furans	Furans	Furans	Furans
	PAHs		PAHs
			1,1,1-Trichloroethane
			Tetrachloromethane
			Trichlorobenzene
			Trichloroethene
			Tetrachloroethene
			Xylenes
	Cadmium	Cadmium	Cadmium
	Lead	Lead	Lead
	Mercury		Mercury
			Arsenic
			Chromium
			Copper
			Nickel
			Zinc

Abbreviations: EDB, 1,2-dibromoethane; PAHs, polycyclic aromatic hydrocarbons.

(*3,9,10*). Battelle no longer produces this database (*11*). Environment Canada has committed resources to estimate total global production and atmospheric loading of POP substances. Reports are now complete for toxaphene and lindane. Reports for other POPs are scheduled in the next 10 years. Wood MacKenzie Company in the United Kingdom, a commercial company, tracks production of the 40 largest chemical companies that account for 80% of the world market. It reports no POP production in 1997 (*12*).

In the United States the volume of chemical production was reported by the International Trade Commission in the "Synthetic Organic Chemicals: U.S. Production and Sales" report (*13*). These data were collected annually for 78 years until the U.S. Congress stopped publication in 1996. There now is no central government reporting mechanism in the United States that reports the volume produced annually in the United States of any given chemical.

Trade. Globally in 1997, of the top 30 pesticide-producing countries, industrial countries accounted for 85.3% of pesticides exported. Developing countries were responsible for 14.7%, a share that is growing (*14*). The PIC Agreement focuses on how to control the trade of POPs. Tools to evaluate the trade of the nine agrochemical POPs have been improved by adoption of Harmonized Tariff Schedules (HTS) between European Union (EU) countries, North America and many other countries involved in world trade (*15*). However, POPs are difficult to track for three reasons:

First, chemicals are grouped into "trade baskets." Tracking specific chemicals is difficult because of the number of substances grouped together. The *Merck Index* (*16*) describes the chemical composition of any chemical in trade and assigns it a Chemical Abstract Service (CAS) Registry Number. Therefore, the number of similar chemicals in this type of basket can fluctuate from year to year.

HCB and DDT are identified in one HTS basket (HTS 290362). However, larger baskets obscure the trade of other POPs. The basket containing aldrin, chlordane, HCH, and mirex (HTS 290359) contains 3 or more other substances. Toxaphene is grouped in a category of 17 substances. Dieldrin and endrin are in a trade basket of 11 substances (HTS 291090). The import/export of mirex alone can be identified in trade from the United States and Canada when the HTS is used at the seven-digit level (HTS 290359.30) but it cannot be tracked in world trade because the HTS is not standardized at the seven-digit level among the EU countries. Although it is difficult to discern when dieldrin is traded among the large number of other chemicals in its HTS category, large obsolete stockpiles of dieldrin have been inventoried by the Food and Agriculture Organization of the United Nations in Africa. For example, Mauritania reports an inventory of 199 tons (*17*). Destruction of these stockpiles is important because of the long-term harmful ecologic effects of POPs.

A second reason POPs are difficult to track is that the HTS delimits some chemicals by use. For example, aldrin, chlordane, and heptachlor (HCH) are identified in their trade basket by chemical composition but are restricted by use only as pesticides. This does not report trade of any of these substances for other possible uses—for example, as chemical intermediaries.

Third, some trade baskets include specific substances and all their commercial uses. For example, DDT and HCB are listed as named substances. The number of chemicals in this basket each year remains the same and includes all uses of the substance. HCB, while listed as one of the UNEP POP agrochemicals, is

rated by the *Hawley's Condensed Chemical Dictionary* (*18*) first as an organic synthesis, next as a seed protectant, and third as a wood preservative. This indicates that HCB is traded as an industrial intermediate product in the largest volume. This may explain why the import/export data in the HTS code does not show a decline in trade in the United States from 1992 to 1998 even after these chemicals were banned for agricultural uses.

It is important to know that only about 25% of the agricultural chemicals are shipped as pure substances and therefore labeled. Most chemicals are shipped as mixes and are not labeled and not tracked by HTS codes (*19*). The import/export data available from HTS codes for POPs, therefore, present only a partial picture of the world trade of these substances.

It appears, however, that consumption of chlorinated hydrocarbons in the United States has continued at about the same level every year from 1989 to 1996—3.4 million pounds. Exports of chlorinated hydrocarbons are reported as 3.9 million pounds in 1994 and 1.7–3.9 million pounds in 1996 (*8*). The United States reports annual consumption at about 2.3–3.7 metric tons (*20*), indicating that mid-sized companies are producing the substance. Persistent production of at least some of the agrochemical POPs is substantiated by trade data from the United States and around the world (*15*).

Focus on the United States

Aldrin and Dieldrin in the Environment

The second part of this review focuses on only two of the POP substances, aldrin and dieldrin, and how they are evaluated within a research establishment in the United States.

In this review, I examine as many data sources as possible across the environmental continuum. Many environmental scientists are working with aldrin and dieldrin and many of their research findings are complementary. In this review I have adapted a graph developed by Lioy as a useful method to separate and describe groups of scientists working on these two substances (*21*). Topic headings use the format of Lioy's graph (Figure 1).

Environmental Science. **Production.** Aldrin and dieldrin were first formulated from a waste product of synthetic rubber, cyclopentadiene. Aldrin, dieldrin, and endrin are cyclodienes made by a chemical process known as the Diels-Alder reaction, hence their names.

Aldrin and dieldrin were discovered by Julius Hyman. Hyman worked for Velsicol Corporation, headquartered in Memphis, Tennessee, and helped discover chlordane. Chlordane was produced in a plant in Marshal, Illinois, from 1944 to the 1980s. In 1947, Hyman left to form his own company, hoping to produce aldrin and dieldrin. However, a court suit followed and Velsicol was found to retain the patent rights for these substances (*23*). In 1948, Shell Chemical bought Hyman and Company and began production of aldrin and dieldrin in its facility in Denver, Colorado. Shell was the only producer until April 1968, when AMVAC, headquartered in Los Angeles, California, also began to produce both substances (*23*).

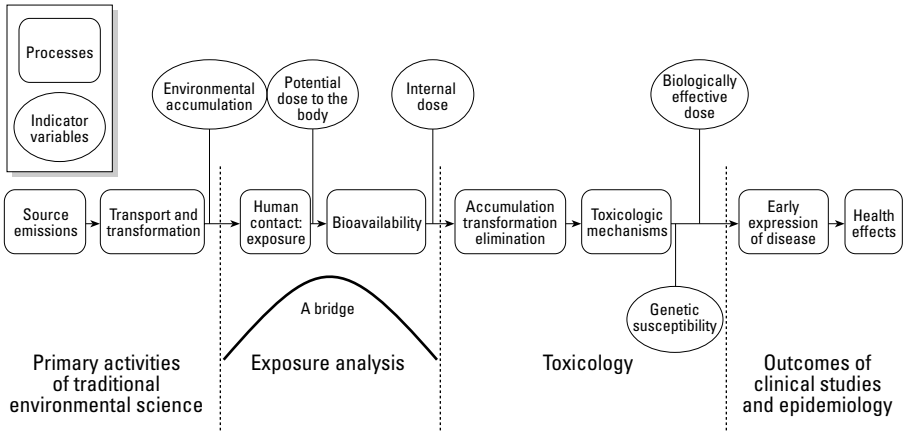

Figure 1. Process continuum from emission of a contaminant to a health effect. Adapted from Lioy (*21*).

The U.S. Tariff Commission did not begin reporting the volume of production of an aldrin group until 1968 when it was included in the annual "Synthetic Organic Chemicals: U.S. Production and Sales" publication (*13*). Although production started at Shell Chemical in 1948, the amounts of aldrin and dieldrin produced could not be reported without revealing company propriety information until 20 years later, when AMVAC began production.

In 1968, the U.S. Tariff Commission report included aldrin and dieldrin in a trade group with chlordane, HCH, terpene polychlorinates, and toxaphene. This is important because by the 1990s the HTS basket had been rearranged. Dieldrin had been separated from this group and placed into another trade basket that included 11 or more other substances—too many to determine when dieldrin was traded.

While estimates of production of aldrin and dieldrin vary, it is probable that their combined production rose to a peak of about 20 million pounds a year in the mid-1960s and then declined (*22*). U.S. domestic use data indicate production was about 90% aldrin and 10% dieldrin (*23*).

The U.S. EPA Pesticide Product Information System on the Internet reports 36 manufacturing and distribution facilities for aldrin and dieldrin (*24*). The U.S. EPA database reports all production at these facilities has been cancelled, except six companies that report they have "transferred" their license. However, combined with data from the *SRI Chemical Economics Handbook* (*8*), the Purdue Silver Platter pesticide CD-ROM (*25*), and the *Farm Chemicals Handbook* (*26*), 65 companies report they at sometime manufactured aldrin or/and dieldrin in the United States. Heavy concentrations of facilities were located in New York. There were 10 in New Jersey and 4 in California (Figure 2).

The U.S. National Library of Medicine reported in 1998 that all U.S. manufacture and use of aldrin and dieldrin had been discontinued (*27*). However, a search of the industry trade literature (*8,24–26*) reveals that 11 companies reported production of aldrin or dieldrin from 1989 to 1999 and 4 companies reported distribution. It is not known if these chemicals are primarily exported or if they are used as chemical intermediaries for other products or only for scientific research (Figure 2, Table 2).

It follows that states in which production facilities are located also have the highest number of hazardous waste sites listed by U.S. EPA on the National Priority List (NPL) for cleanup. States that have 7–21 NPL sites for aldrin and dieldrin are in New York, Washington, and Florida where manufacturing facilities were located and in Ohio, Indiana, and Michigan where heavy use was reported (*10*).

Agricultural use in pounds. Aldrin/dieldrin ranked second—after DDT—among agricultural chemicals used in the United States in the 1960s. The distribution maps show aldrin use was most concentrated in the Midwest; dieldrin was used more heavily in the South, west coast states, and Massachusetts. Estimates of use for dieldrin and aldrin were made by the U.S. Geological Survey (USGS) by calibrating crop harvests data from 1978 and typical pesticide use rates from 1966 (*28*). USGS does not list these data on its website, but U.S. EPA developed the data for its "Great Lakes Binational Toxic Strategy Draft

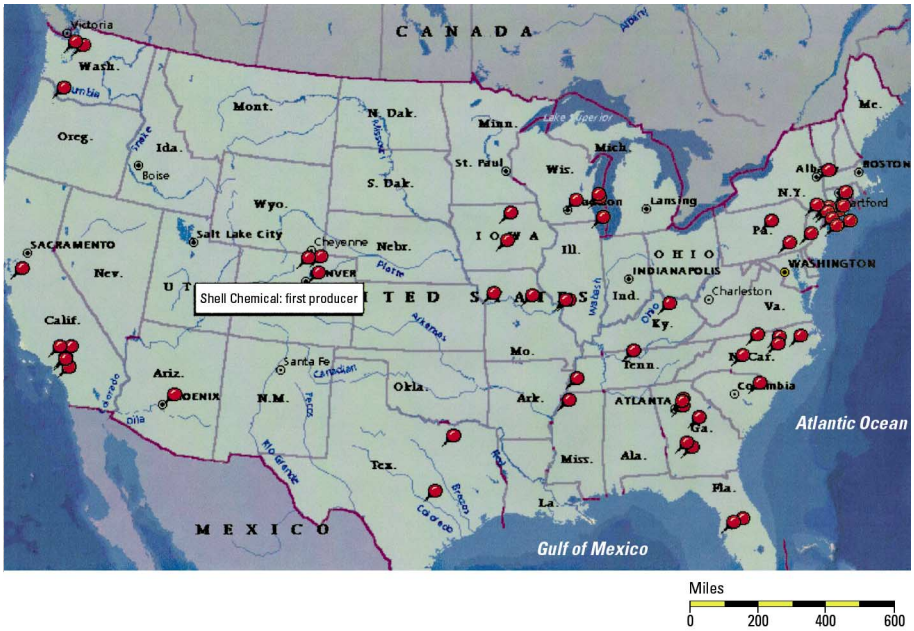

Figure 2. Aldrin and dieldrin manufacturing facilities in the United States.

Table 2. Aldrin or dieldrin: a comparison of four pesticide directory databases.[a]

U.S. EPA-Registrants *http://www.cdpr.ca.gov/cgi-bin/epa/comp.pl?pccode* (*24*) All companies reported as inactive	Purdue CD-ROM (*25*) Companies reported as inactive or transferred	*Farm Chemicals Handbook* (*26*) Active producers, distributors on the dates listed 1970, 1969, and 1989–1996	*Chemical Economics Handbook*-SRI (*8*) Active producers, distributors on the dates listed 1982, 1985, 1992–1999
			Accurate Chem. and Sci. 300 Shames Dr. Westbury, NY 11590 Tel 516-333-2221 A/D (high purity) Distributor only 1982,1985, 1996-1999
			614 San Diego, CA 92103 Tel 619-235-9400 Sales
			Accustandard Inc. 25 Science Park New Haven, CT 92112-6129 A/D (high purity) Sales/ship 1998
			Aldrich Chemical 1001 West St. Paul Ave. Milwaukee, WI 53233 Tel 414-273-4979 D Bulk; bulk manuf/sales 1992–1999
			Alltech Associates 2051 Waukegan Rd. Deerfield, IL 60016 Tel 312-948-8600 A/D manuf 1982
AMVAC Chemical Corp. 2110 Davie Ave. Commerce, CA 90040-1706 A manuf 43% (Registration 1986–1987) D manuf 18.7% Royal brand dieldrin (Registration 1986–1989)	AMVAC Chemical Corp. 2110 Davie Avenue Commerce, CA 900401706 Royal brand dieldrin 18.7% A/D manuf	AMVAC Chemical Corp. 4100 E Wash. Blvd. Los Angeles, CA 90023 Tel 213-264-3910 A manuf 1969, 1992	
Arizona Chem C6 PO Box 21537 Phoenix, AZ 85036 A manuf (Registration 1968–1986)			

(Continued)

Table 2. Continued.

U.S. EPA-Registrants *http://www.cdpr.ca.gov/cgi-bin/epa/comp.pl?pccode* (*24*) All companies reported as inactive	Purdue CD-ROM (*25*) Companies reported as inactive or transferred	*Farm Chemicals Handbook* (*26*) Active producers, distributors on the dates listed 1970, 1969, and 1989–1996	*Chemical Economics Handbook*-SRI (*8*) Active producers, distributors on the dates listed 1982, 1985, 1992–1999
Bell Chemical Co. PO Box 59267 Dallas, TX 75229 Tel 214-484-5135 D manuf 18.6% Belco	Bell Chemical Co. Dallas, TX 75229 Belco 18.6% D manuf D distributor 1969 Registration 1958–1989)	Belco Resources Bell Chemical Co. Dallas, TX 75229	
			Calif Bionuclear Corp. 7654 San Fernando Rd. Sun Valley, CA 91352 1,2,3,4,10-C14 A/D manuf/sales 1982–1985
CEN/EX Land O'Lakes PO Box 536 Hampton, IA 50441 Tel 515-562-2500 (Registration 1965–1989) A Formulator			
Chem-nut, Inc. PO Box 3706 Albany, GA 31706 A manuf Master brand AA 23.2–42.8% (Registration 1981–1984) D manuf Master brand spray 14 18.6% (Registration 1981–1987)	Chem-nut, Inc. Albany, GA 31706 Master brand spray 18.6% A/D manuf		
			Chempar Chem Co. 60 East 42nd St. New York, NY 10017 Tel 212-687-3990 A/D Sales 1982
		Chem. Service, Inc. PO Box 3108 660 Tower Rd. 12 West Chester, PA 19381-3108 Tel 610-692-3026 A/D 15 (high purity) manuf/sales/ship 1992	Chem. Service, Inc. PO Box 3108 660 Tower Rd. West Chester, PA 19381-3108 Tel 610-692-3026 A/D (high purity) manuf/sales/ship 1992–1999
Coastal Chem. C6 PO Box 856 Greenville, NC 27834 (Registration 1966–1987) A formulator			
			Crescent Chem. Co. 1324 Motor Parkway Hauppauge, NY 11788 Tel 516-348-0333 A (high purity) sales/shipping 1985–1999
Darling & Co. 322 N. Commercial – Suite #5 PO Box 87 Eagle Grove, IA 50533 Drewclad mothproofer 6.67%A Formulator 1%			
Drew Industrial Div. One Drew Plaza Ashland Chemical Co. Divi Boonton, NJ 07005-1924 D manuf Drewclad mothproofer 6.67% (Registration 1969–1985)	Drew Industrial Div. One Drew Plaza Ashland Chemical Co. Divi Boonton, NJ 07005-1924 Drewclad mothproofer 6.67% D manuf		
Farmingdale Garden Labs 136 Verdi St. Farmingdale, NY 11735 D manuf 18.6% Dieldrin termite proofer (Registration 1973–1985)	Farmingdale Garden Labs 136 Verdi St. Farmingdale, NY 11735 Dieldrin termite proofer 18.6% D manuf transferred		

(Continued)

Table 2. Continued.

U.S. EPA-Registrants *http://www.cdpr.ca.gov/cgi-bin/epa/comp.pl?pccode* (*24*) All companies reported as inactive	Purdue CD-ROM (*25*) Companies reported as inactive or transferred	*Farm Chemicals Handbook* (*26*) Active producers, distributors on the dates listed 1970, 1969, and 1989–1996	*Chemical Economics Handbook*-SRI (*8*) Active producers, distributors on the dates listed 1982, 1985, 1992–1999
Farmland Industries 3315 N. Oak Trafficway Kansas City, MO 64116 Tel 816-459-5479 A formulator 20% (Registration 1957–1987)			
			Fluka Chem. Corp. 255 Oser Ave. Hauppague, NY 11787 Tel 516-273-0110 D sales/shipping 1985, 1992
FCX, Inc. PO Box 2419 Raleigh, NC 27642 A Formulator			
Gabriel Chemical Ltd. 204 21st Ave., PO Box 2138 Patterson, NJ 07509 Also 333 Hanover, PA 17331 A Formulator (Registration 1969–1989) D manuf 18.0% Registration 1970–1984)			
Garden Care by Farmingdale Ltd. Greeley, CO 80632 Tel 303-353-0611 D manuf 18.6% EC dieldrin termite	Garden Care by Farmingdale Ltd. Greeley, CO 80632 EC dieldrin termite 18.6% D manuf (Registration 1985–1987)		
Gregory B. Crampton, Trustee 100 St. Albans Dr., Ste. 200 Raleigh, NC 27619 A manuf (Smith Douglass Seed/rice treatment, fertilizer) (5–43.3%) (Registration 1962–1989) D manuf (Smith Douglass 24.6%) (Registration 1967–1988)	Gregory B. Crampton, Trustee F 100 St. Albans Dr., Ste. 200 Raleigh, NC 27619 Smith Douglass dieldrin 24.6% A/D manuf transferred		
Gold Kist, Inc. PO Box 2210 Atlanta, GA 30301 Registration 1971–1983) A Formulator 30%			
HACO, Inc. PO Box 7190 Madison, WI 53707 Tel 608-221-6200 Dieldrin termite control 18.6% D manuf (Registration 1982–1985)	HACO, Inc. PO Box 7190 Madison, WI 53707 Dieldrin termite control 18.6% D manuf transferred	Hopkins Ag. Chem. Co. 537 Atlas Ave, PO Box 584 Madison, WI 53707 Randolf, WI (Plant) D manuf 1970	
IBC Manuf. Co. 416 E. Brooks Rd. Memphis, TN 38109 Tel 901-344-5316 (Registration 1959–1989) A manuf 42.5%			
			ICN Biomedicals, Inc. K&K Rare/Fine Chem. PO Box 5023 Costa Mesa, CA 92626 D Sales 1999 121 Express St Plainview, NY 11803 D sales/shipping 1985

(Continued)

Table 2. Continued.

U.S. EPA-Registrants *http://www.cdpr.ca.gov/cgi-bin/epa/comp.pl?pccode* (*24*) All companies reported as inactive	Purdue CD-ROM (*25*) Companies reported as inactive or transferred	*Farm Chemicals Handbook* (*26*) Active producers, distributors on the dates listed 1970, 1969, and 1989–1996	*Chemical Economics Handbook*-SRI (*8*) Active producers, distributors on the dates listed 1982, 1985, 1992–1999
			Icon Services, Inc Old Kings Hwy. Mt. Marion, NY 12456 Tel 914-246-1802 manuf/sales 1999 19 Ox Bow Lane Summit, NJ 07901 Tel 908-273-0449 D 13CD sales 1999
			Lachat Chem., Inc. 10500 N, Point Wash. Rd. Mequon, WI 53092 Tel 414-241-3872 D manuf/sales 1982–1985
Landia Chem. Co. PO Box 6884 Lakeland, FL 33807 Tel 813-647-3608 (Registration 1980–1985) A manuf (Fasco liq. 42.55%)			
Los Angeles Chem. Co. South Gate, CA 90280 D manuf LACCO Dieldrin mulsion 19.5% (Registration 1974–1986)	LACCO Los Angeles Chem. Co. South Gate, CA 90280 Dieldrin mulsion 19.5% D manuf	Los Angeles Chem. Co. 4545 Ardine St. South Gate, CA 90280 Tel 213-583-4761 D manuf 1970 Marshall Thomas Washington State A/D sales 1989–1993	
			Merck Sharp & Dohme Isotopes PO Box 2951 Terminal Annex Los Angeles, CA 90051 Tel 213-723-9521 A/D 13CD manuf/sales/shipping 1992
MFA Oil Co. 200 S. 7th St. #519 Columbia, MO 65201 Tel 712-246-2150 D manuf dieldrin 18.7% (Registration 1973–1987)	MFA Dieldrin 18.7% MFA Oil Co. 200 S. 7th St. Columbia, MO 65201 D manuf	Missouri Farmers Assoc. 201 S. 7th St. (plant) Columbia, MO 65202 Tel 443-873 Boone City, MO 65202 (plant) D Manufacture 1969, 1970	
Micro-Flo Co. Memphis, TN 38117-2099 Tel 901-432-5000 (Registration 1985–1987) A manuf 42.55%			
Miller Chemical & Fertilizer Co. Pratt-Gabriel Division PO Box 3 Hanover, PA 17331 D manuf dieldrin termite proofer 18% (Registration 1970–1984)	Miller Chemical & Fertilizer Co. Pratt-Gabriel Division Hanover, PA 17331 Dieldrin termite proofer 18% D manuf transferred	Miller Chemical & Fertilizer Co. (plant) PO Box 311 Hanover, PA 17331 D manuf/formulator 1969,1970 Baltimore, MD D Formulator 1969	
	McLean's NAMCO Chem. Inc. 2256 Junction Ave. San Jose, CA 95131 A manuf		
			NCI–CHM Carcinogen Ref. Standard 425 Volker Blvd. Kansas City, MO 64110 Tel816-753-7600 A/D (high purity) manuf/sales/ship 1996–1999

(Continued)

Table 2. Continued.

U.S. EPA-Registrants *http://www.cdpr.ca.gov/cgi-bin/epa/comp.pl?pccode* (*24*) All companies reported as inactive	Purdue CD-ROM (*25*) Companies reported as inactive or transferred	*Farm Chemicals Handbook* (*26*) Active producers, distributors on the dates listed 1970, 1969, and 1989–1996	*Chemical Economics Handbook*-SRI (*8*) Active producers, distributors on the dates listed 1982, 1985, 1992–1999
			NSI Environ. Solutions Inc. PO Box 12313 Research Triangle Park, NC 27709 Tel 919-544-0414 A/D (high purity) manuf/sales/ship 1992–1999
			Pathfinders Lab Inc. 11542 Fort Mims Dr. St Louis, MO 63146 Tel 314-569-0681 A Sales 1985
	PBI/Gordon Corp. PO Box 014090 Kansas City, MO 64101 Dieldrin 15E D manuf		
Perk Products and Chemical Co. PO Box 100585 Nashville, TN 37224 Tel 615-242-6157 D manuf Perkerson's final notice 17% (Registration 1969–1987)	Perk Products and Chemical Co. Nashville, TN 37224 Perkerson's final notice 17% D manuf/sales	Perk Products and Chemical Co. 1338 Lewis St. Nashville, TN 37210 D manuf/sales 1969	
Prentiss Inc. C.B. 2000 Floral Park, NY 11001-2000 Tel 516-326-1919 (Registration 1957–1985) A manuf (Pentox 2.48%) D manuf (Pentox 20–50%) (Registration 1955–1985)	Prentiss Inc. C.B. 2000 Floral Park, NY 11001-2000 Pentox wettable power 50% Dieldrin oil soluble 20% A/D sales/manuf	Prentiss Inc. C.B. 2000 Floral Park, NY 11001-2000 Newark, NJ (plant) A/D manuf	
			Protocal Analytical Supplies Inc. 472 Lincoln Blvd. Middlesex, NJ 08846 Tel 732-627-0500 A/D (high purity) manuf/sales/ship 1996–1999
Radian Intl. PO Box 201088 Austin, TX 78720-1088 Tel 512-238-9974 A/D(high purity) manuf/sales/ship 1996–1999			
Red Panther Chem. Co. PO Box 326 Clarksdale, MS 38614 D manuf Smith Douglass 24.6% (Registration 1988–1989)	Red Panther Chem. Co. PO Box 326 Clarksdale, MS 38614 Smith Douglass 24.6% D manuf	Red Panther Brand COAHOME Chem. Co. Inc. PO Box 550 (plant) Clarksdale, MS 38614 Plant also in Illinois D manuf/sales 1969	
Scallop Corp. One Rockfeller Plaza New York, NY 10020 (Registration 1984–1987) A manuf (Terminex 42.8%) D manuf dieldrin termite 18.6% (Registration 1985–1989)	Scallop Corp. One Rockfeller Plaza New York, NY 10020 Dieldrin termite control 18.6% A/D manuf		
SELCO Supply Co. PO 1286 419 18TH St. Greeley, CO 80632 D manuf SELCO 18.6% (Registration 1966–1985)	SELCO Supply Co. 419 18th St. Greeley, CO 80632 SELCO 18.6% D manuf	SELCO Supply Co. 419 18th St. Greeley, CO 80632 1 Collins Ave. & RR Yd (Plant) PO Box 277 Eaton, CO 80615 2 Severance, CO D manuf	

(Continued)

Table 2. Continued.

U.S. EPA-Registrants *http://www.cdpr.ca.gov/cgi-bin/epa/comp.pl?pccode* (*24*) All companies reported as inactive	Purdue CD-ROM (*25*) Companies reported as inactive or transferred	*Farm Chemicals Handbook* (*26*) Active producers, distributors on the dates listed 1970, 1969, and 1989–1996	*Chemical Economics Handbook*-SRI (*8*) Active producers, distributors on the dates listed 1982, 1985, 1992–1999
			Serva Biochem. 50 A & S Dr. Paramus, NJ 076552 Tel 201-967-8858 A/D manuf/ship/sale 1992–1994
Shell International Chem. Co. Ltd. London S.E. 17 PG Denver, CO A manuf D manuf Shell Tech dieldrin (Registration 1984–1989)	Shell International Chem. Co. Ltd. 1025 Connecticut Ave. Washington, DC 200036 Pratt's termite 18% Shell Tech dieldrin na% A/D manuf	Shell Chem. Co-Agric. Div. PO Box 2171 (plant) Denver, CO 80201 A/D manuf/sale 1969	
			Sigma Chemical Co. PO Box 14508 (63178) 3050 Spruce Street St. Louis, MO 63103 Tel 314-771-5765 D manuf/sales/ship 1996–1999
Southern Agricutural Chemicals Rt. 1, Cades, SC 29518 (Registration 1973–1989) A manuf 43.4% D manuf Royal brand dieldrin 18.7% (Registration 1973–1986)	Southern Agricultural Chemicals Rt. 1, Cades, SC 29518 Royal brand dieldrin 18.7% D manuf/transferred	Southern Agricultural Chemicals Rt. 1, Cades, SC 29518 PO Box 527 Kingstree, SC 29556 (plant) Suspension-type liq. fertilizer D manuf/sales 1970	
Southern Mill Creek 10008 Dale Mabry #121 Tampa, FL 33618 Tel 813-960-8333 (registratiion 1974-91) A manuf (SMCP Aldrin 43.4%)			
Stephenson Chem. Company Inc. PO Box 87188 College Park, GA 30337 A manuf 46.3% (Registration 1959–1989) D manuf (Stephenson 17.84%) (Registration 1975–1987)	Stephenson Chem. Company Inc. PO Box 87188 College Park, GA 30337 Stephenson 17.84% A/D manuf	Stephenson Chem. Company Inc. 2444 West Point Rd. (plant) PO Box 188 College Park, GA 30337 D manuf/sales 1969	
Stevens Indus Inc. N. Main St., PO Box 272 Dawson, GA 31742 A manuf Masters brand 25% (Registration 1967–1989) D manuf Master brand TermiteKill 18.62% (Registration 1960–1981)	Stevens Indus. Inc. (Plant) N. Main St., PO Box 272 Dawson, GA 31742 Master brand D manuf/sales 1969, 1970		
			Supelco, Inc. Supelco Park Bellefonte, PA 16823-0048 Tel 814-356-3044 A/D(high purity) manuf/sales/ship 1996–1999 6 other overseas manuf sites
Terminix Div. of Cook Indus., Inc. PO Box 17167 Memphis, TN 38117 A manuf 42% (Registration 1951–1986) D manuf 0.25–16.6% (Registration 1951–1986)	Terminix Div. of Cook Indus., Inc. PO Box 17167 Memphis, TN 38117 Termastic 0.25% Terminix 17.6% D manuf	Terminix Wood Treating and Cont. Co. 920 Sheridan St. Honolulu, HA 96814 D Formulator 1970	
			TCI America 9211 N. Harborgate St. Portland, OR 97203 Tel 503-283-1681 D manuf/sales/ship 1996–191999 Also manuf/ship from Japan

(Continued)

Table 2. Continued.

U.S. EPA-Registrants *http://www.cdpr.ca.gov/cgi-bin/epa/comp.pl?pccode* (*24*) All companies reported as inactive	Purdue CD-ROM (*25*) Companies reported as inactive or transferred	*Farm Chemicals Handbook* (*26*) Active producers, distributors on the dates listed 1970, 1969, and 1989–1996	*Chemical Economics Handbook*-SRI (*8*) Active producers, distributors on the dates listed 1982, 1985, 1992–1999
Tobacco States Chemical Co. 130 Trafton St. Lexington, KY 40504 A manuf 41.22% (Registration 1975–1986) D manuf 17.6% (Registration 1964–1983)		Tobacco States Chemical Co. (plant) 130 Trafton St. Lexington, KY 40501 D Formulator 1969 Plants also in Louisville, KY; Nashville, TN; Evansville, IN	
Triangle Chemical Co. Box 4528 Macon, GA 31208 A manuf D termite 18.7% (Registration 1959–1987) D manuf 18.7% (Registration 1952–1987)	Triangle Chemical Co. PO Box 4528 Macon, GA 31208 Triangle Chemicals 18.7% A/D manuf	Triangle Chemical Co. 206 Lower Elm St. Macon, GA 31208 Triangle Chemicals 18.7% D Formulator 1969	
			Tridom Chem., Inc. 255 Oser Ave. Hauppague, NY 11787 Tel 516-273-0110 D Sales 1982
Van Waters & Rogers, PO Box 34325 Seattle, WA 98124-1325 Tel 206-889-3911 NAMCO 18.6% D manuf (Registration 1976–1987)	Van Waters & Rogers, Inc. Seattle, WA 98124-1325 NAMCO 18.6% D manuf	Van Waters & Rogers, 4000 First Ave. Seattle, WA 981341325 D Formulator 1969 Plants also in Anchorage, AK; Phoenix, AZ; Brisbane, CA; Fresno, CA; Los Angeles, CA; Denver, CO; Honolulu, HI; Boise, ID; Portland, OR; Independence, OR; Albany, OR; Dallas, TX; Salt Lake City, UT; Seattle, WA; Spokane, WA; Bruce, WA	
			Wako Chem. 1600 Bellwood Rd. Richmond, VA 23237 Tel 800-992-WAKO D (high purity) A/D manuf/sales 1992, 1996–1999
Wood Protection Products, Inc. 650 State St. Charlotte, NC 28208 Tel 704-372-6790 D manuf for dieldin 18.6% (Registration 1974–1989)	Wood Protection Products, Inc. 650 State St. Charlotte, NC 28208 For dieldrin 18.6% D manuf		
			VWR Scientfic Products 1310 Goshen Parkway West Chester, PA 19380 Tel 610-431-1700 A/D Sales/shipping 1996–1999 8 U.S. locations

Abbreviations: A, aldrin; D, dieldrin; manuf, manufacture.

Report," December 1998 (*10,29*). U.S. EPA air monitoring data (*10*) record how aldrin and dieldrin volatize off fields and are carried in wind patterns (Figure 3).

Aldrin was used as a soil poison for the control of corn rootworms, cutworms, and wireworms. Use on farms was mainly allocated to corn—96.6% in 1966 and 88% in 1971. It was used in the greatest concentrations in the corn belt states; Iowa and Illinois alone accounted for 59% of the total acreage treated in 1971. Aldrin was applied to Iowa soils at the rate of 5–6.5 million pounds between 1961 and 1965. It was reduced to 2 million pounds from 1968 to 1973 (*30*). States outside the Midwest accounted for only 2.7% of the aldrin used on corn in 1966, declining to 0.5% in 1971 (*23*).

Dieldrin had many of the same uses as aldrin but was more expensive to produce. Production of dieldrin was about 10% that of aldrin. It was recommended for use on approximately 90 crops, principally corn, hay, wheat, rye, barley and oats, and orchards and vegetables. In the South it was used on tobacco, cotton, and citrus crops. More than 50% of the dieldrin produced in 1964 was used for pest control instead of agriculture. This included soil application for termite control and mothproofing during wool carpet and clothing manufacturing. These uses significantly contributed to long-term indoor air pollution. Pest control uses also included control of harvest and fire ants and for aerial spraying of spruce budworm and gypsy moths.

Dieldrin distribution patterns are different from those for aldrin. The Northeast, in 1966, used proportionally more dieldrin than

the rest of the United States. The Northeast accounted for only 0.2% of national aldrin sales but 6.1% of national dieldrin sales for crops (*23*). Cherry orchards in Indiana also accounted for the use of large amounts of dieldrin. Urban and mixed-use areas in the Southern States had high use levels of dieldrin for termite control and in home and lawn products.

Environmental accumulation. **Soil.** Aldrin and dieldrin bind to the soil. Aldrin is more volatile and readily degrades to dieldrin in the environment. The rate at which aldrin degrades to dieldrin has been estimated. When aldrin is applied to silty loam soil, the amount detectable in 1.7 years will have declined by 25% of the amount applied. Aldrin is estimated to have a half-life in soil of 1.5–5.2 years, depending on the composition of the soil. More than 56% of the original weight of aldrin converts to dieldrin; about 19% of the original aldrin weight disappears. This loss has not been accurately accounted for (*23*).

Dieldrin degradation in the environment appears to be a function of the concentration of dieldrin in the soil. Beyer and Gish (*31*) determined that for dieldrin used at the rate of 0.6 kg/ha the half-life was an average of 2.6 years, at 2.2 kg/ha the half-life averaged over 4.1 years, and at 9.0 kg/ha the half-life was 12.5 years. Dieldrin, in contrast, has a lower volatility (vapor pressure = 1.78×10^7 mm Hg at 20°C). Both aldrin and dieldrin are absorbed into the food chain. Residues remain in the soil for a long period, as contaminated plant and animal materials are added to the topsoil. Dieldrin is retained in the fatty materials of sewage sludge and in fish emulsions used as fertilizers. Topical soil application of these materials makes dieldrin available to grazing animals, who ingest some soil when they crop grass.

Water. Aldrin and dieldrin are hydrophobic and do not dissolve easily in water. Dieldrin especially has low solubility in water (186 µg/L at 25–29°C). It is not dissolved by water passing through the soil, but is very soluble in fats, waxes, and oils (*32*).

However, a 1993 study (*28*) shows that dieldrin was more frequently detected than the 26 other insecticides studied at 1,016 sites located in 50 of the major river basins of the United States. Sampling from 50 large U.S. river-basin networks indicated that 90% of water and fish samples contained organochlorines; DDT and dieldrin were the most prevalent of these (*33*). A USGS database lists 50 U.S. watersheds where dieldrin is found (*33*) (Figure 4).

Limited sediment core samples available from the Great Lakes in the United States indicate dieldrin contamination began in the 1940s and generally peaked around the 1970s. There has been a decrease in detected levels in recent years (*10*). USGS has performed core sediment studies on more than 70 lakes in order to examine the historical record of pesticide use, but these data are not yet available on the Internet.

Transport, transformation, and environmental deposition and fate of dieldrin. **Air transport.** The atmospheric photooxidation half-life of aldrin is estimated to be between 55 min and 9.1 hr. The photooxidation half-life for dieldrin is much longer, an estimated range of 4–40.5 hr.

Scientific estimates about how aldrin and dieldrin travel in the environment can be made using air modeling and ice and soil cores. Work in this field was summarized in 1993 by Wania and Mackay (*34*). They report how organochlorines present in the atmosphere "condense" into soil, water, aerosols, snow, and ice as the temperature falls. This evaporation and condensation cycle is called "grasshoppering" and allows the chemicals to travel long distances. In 1975, Goldberg (*35*) described the "global distribution" of organochlorines through the atmospheric transfer of DDT from continents to oceans. Ottar (*36*), in 1981, described POPs deposition and re-emission rates as, "a systematic transfer of the more persistent compounds from warmer to colder regions . . . to accumulation of these substances in the temperate and arctic regions." This work was supported by contaminant work monitoring the relative volatility of organochlorines and mapping how they are found in higher concentrations near the source of pollution, with declining concentrations in the air, atmospheric deposition, fish, and seal samples as they are sampled into more remote and colder regions. Core samples of lake sediments, and lakes in higher altitudes tract the historical movement of POPs to cooler temperatures. Dieldrin shows up at the highest levels in lakes 75 degrees North compared to more southern lakes beginning at 49 degrees (*37*). Patton et al. (*38*) investigated ice cores and found that freshly fallen snow had 2–6 times

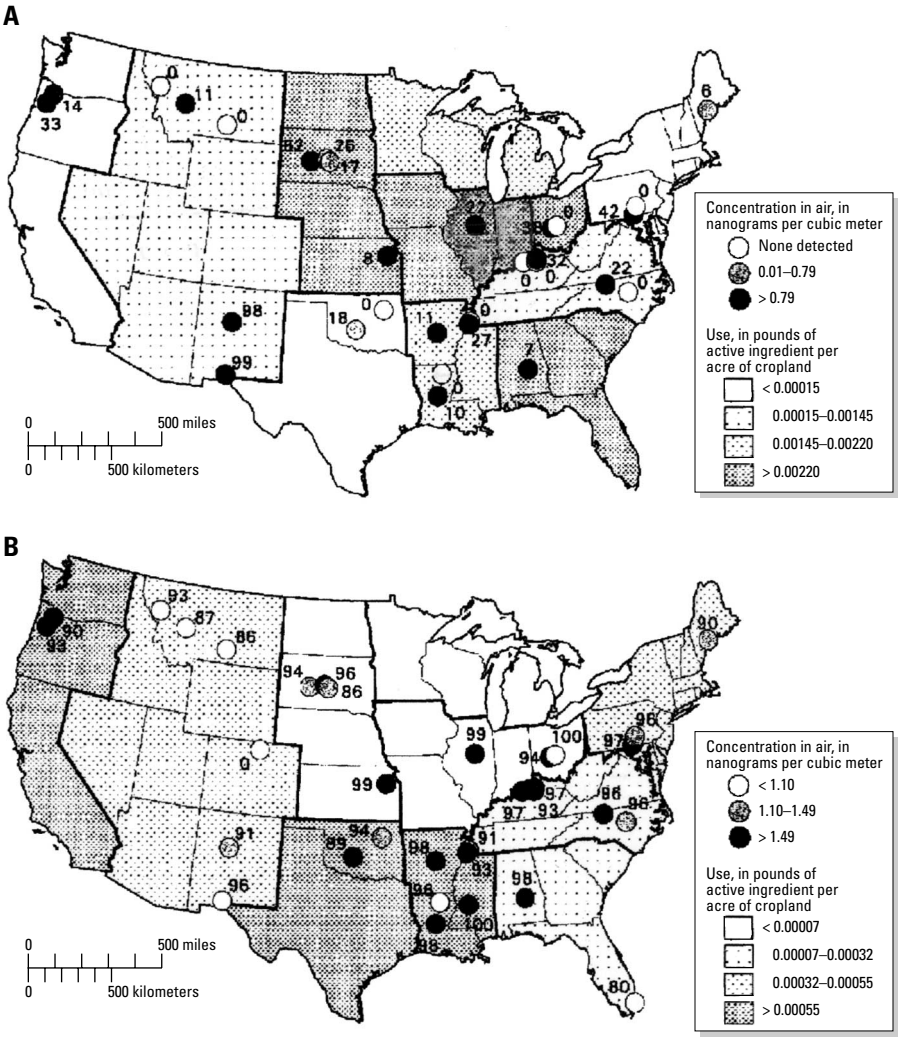

Figure 3. (*A*) Aldrin and (*B*) dieldrin measures by pounds per acre of crop land and location of air monitoring stations. Maps reproduced from the U.S. EPA (*9*).

higher POPs than subsurface snow. This suggests that during the summer POPs revolatize from the snow as temperatures warm up or may be released by changes in the physical structure of the older snow. Dieldrin among other organochlorines are now found as a supersaturated slick on the surface waters in the Arctic (*39*). Wania and Mackay (*34*) also reported the "ecotoxicology" of colder regions and the Arctic:

> Poor mixing of the upper layers of the Arctic Ocean due to a strong halocline (*40*) may increase the availability of the toxic compounds to marine mammals. The lack of soil leaching over permafrost and the low precipitation rates may reult in increased exposure to browsing terrestrial animals (*41*). During snowmelt a sudden release of stored-up pollutants in a shock wave may occur (*42*), coinciding with an especially sensitive phase of the aquatic life cycle.

As dieldrin levels decrease in the United States, they are appearing in increasing concentrations in the colder Arctic areas. The atmospheric data track how dieldrin travels very long distances from the source where it is applied. The deposition of aldrin and dieldrin can accumulate deposits in remote areas thought to be pristine.

The U.S. EPA now has an extensive air monitoring network; however, less than a quarter of the stations report readings for organochlorines (*43*). The Integrated Atmospheric Deposition Network (IADN) reports on their website air monitoring information of POPs by chemical on a seasonal basis (*44*). These locations are seen in Figure 5. Air monitoring stations reporting high levels of aldrin and dieldrin are seen in Figure 3. Unfortunately, New York, New Jersey, and California do not publicly report the air monitoring detection levels of aldrin or dieldrin levels.

Air particle monitoring estimation of dieldrin elimination from the atmosphere. Following current trends, dieldrin and aldrin are projected to disappear from U.S. air particle monitoring networks around 2030, almost a century after they were first used (*44,45*). It is estimated that DDT, dieldrin, chlordane, and HCB will disappear from the atmosphere and air monitoring samples around the Great Lakes between 2010 and 2060.

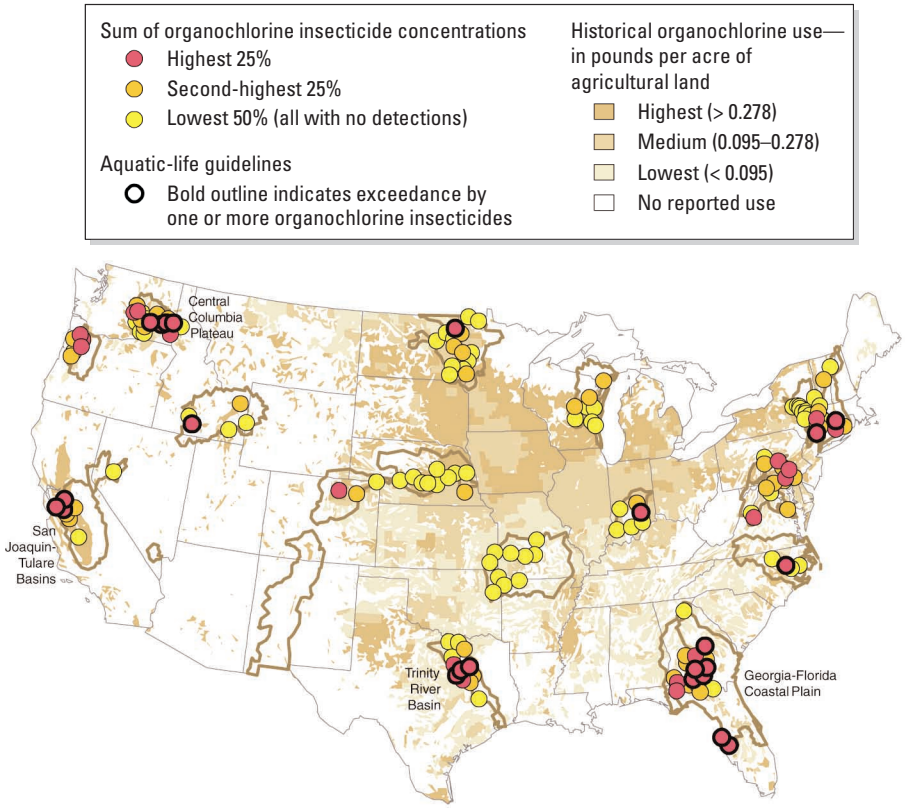

Figure 4. National ranking of organochlorines in bed sediment. Map is reproduced from USGS (*33*).

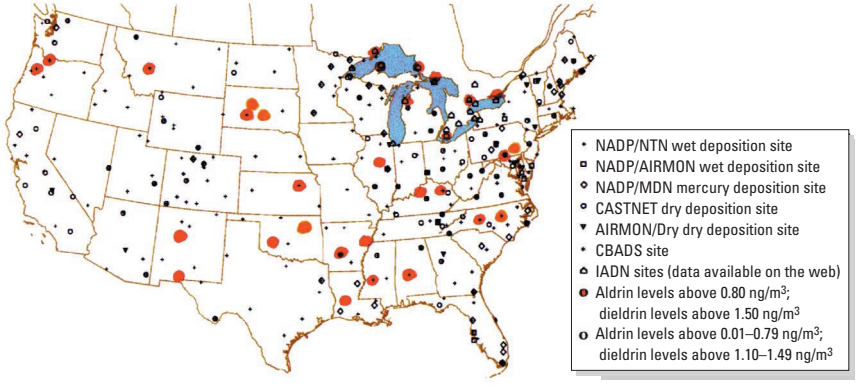

Figure 5. Monitoring sites of ambient air and deposition monitoring networks measuring pollutants of concern. Data reproduced from U.S. EPA (*43*).

Exposure Analysis

Bioavailability. **Bioaccumulation in species.** Environment Canada provides a good definition for terms used in this section (*46*). *Bioaccumulation* refers to the uptake of a given substance directly from water or through the consumption of food containing the substance, whereas *bioconcentration factor* (BCF) refers only to the uptake from water. *Biomagnification factor* (BMF) refers to how a contaminant is absorbed by small organisms and increases as it progresses up the food chain.

In 1997, researchers in the Netherlands conducted a study ranking dieldrin bioaccumulation in certain species (*47*). These data were combined with exposure data from the World Health Organization (WHO) and estimates of acute toxicity, which is a useful way to measure the presence of aldrin and dieldrin in the environment (*47,48*) (Table 3).

Species with high BMFs are amphibians, osteichtyes, phyllopoda, insects, birds, marine mammals, mink, otters, and earthworms (*47,48*) (Table 3). Much work remains to *a*) identify contaminant levels for other species, *b*) determine the most appropriate tissues for measuring residues, *c*) record ages, and *d*) provide more coordination among species.

Also, there are limited data on the bioaccumulation of dieldrin within specific species on which to base timeline trends and evaluations to determine if contamination levels are declining. This information is also needed to evaluate how specific contaminants might affect reproduction and contribute to

declining population groups. The U.S. EPA AQUIRE database (*49*) could be made much more accessible if it provided a megastudy index in which studies are grouped by contaminant and specie and ranked bioconcentration levels are being reported. The USGS Wildlife Research Center in Patuxant, Maryland, has already began to do this. This information would also be improved if U.S. government data could be linked to the WHO website. At present there are few ways to map areas of dieldrin concentration across species and to build on interrelationships.

Plants. Dieldrin is readily absorbed into the pulp of vegetables such as squash, melons, cucumbers, and soybeans. In the United States in 1997, dieldrin was found in 77% of the frozen winter squash sampled (*50,51*). Not much is known about bioaccumulation in other plants such as plankton or its role in dieldrin bioaccumulation in fish. Also, little is known about dieldrin levels in other plants eaten by animals in the wild.

Microinvertebrates—insects. Aldrin and dieldrin were formulated to reduce populations of insects. Laboratory tests show that midges are very sensitive to dieldrin as well as five other organochlorines and are good indicators of sediment toxicity (*52*). There is increasing research on how various insect groups have become more resistant to the toxicity of these substances. Work is now being conducted at the Texas A & M (Commerce, Texas) and the University of Arizona (Tempe, Arizona).

Earthworms. Worms readily absorb dieldrin. Studies (*53*) monitoring the half-lives of dieldrin in soils and concentrations in earthworms found that after 20 years of exposure to dieldrin, levels of contamination in earthworms were closely related to the soil in which they lived. Diedrin decreased from 68 to 5.6 ppm in 20 years, with 8% of the dieldrin remaining in the soil after 20 years. This indicates how long dieldrin remains biologically available to wildlife food chains. It is possible dieldrin contamination levels in these small animals can provide valuable low-tech indicators to identify contaminated soils for gardeners planting bioaccumulating food crops or to check historical use of termaticides around the foundations of houses.

Mussels. Aldrin is reported by Environment Canada to have a BCF of 350–44,600. Detection of dieldrin in mussels usually indicates a direct source of contamination or the presence of a high level dieldrin in local sediments. The National Oceanic and Atmospheric Administration (NOAA) created the National Status and Trends Program in 1984 to monitor the quality of the marine environment. From 1986 to 1993, mussels were collected from 300 coastal sites, and a declining trend of 0.810% for dieldrin concentrations in mollusks was noted (*54*). Marine sites with persistently high levels of dieldrin are near urban areas. Other marine sites are in Richmond, California, and along the Texas and Louisiana Gulf coasts, Maryland, and New Jersey. Freshwater dreissenid mussels from 21 sites in the Great Lakes all indicated dieldrin contamination (*55,56*) (Figure 6, Table 4).

Fish. Fish have among the highest bioaccumulation rates for dieldrin. Environment Canada reports a BCF potential for fish of over 10,000. In freshwater fish BCFs vary depending on species from 2,385 to 68,286 (*57*). A national survey by the U.S. Fish and Wildlife Service (*58*) reported a dieldrin geometric concentration level in whole fish of 40 µg/kg. A study by the USGS (*33*) of dieldrin and 15 other organochlorines in streambed sediments and fish found that fish were more highly contaminated and at higher frequencies than adjacent soil samples. Dieldrin is detected in fish at 50% of the sites sampled; 30% of the sites exceeded human health guidelines; and 19% of the sites exceeded the limits of New York guidelines for protection of fish-eating wildlife.

In 1998, NOAA conducted a meta-analysis of fish and sediments at 50 sites along the Pacific coast of the United States. It found, again, fish bioconcentrate organochlorines at higher rates than contaminants found in sediments; data from 1984 to 1990 showed no consistent temporal trends. Dieldrin contamination levels in fish livers measured by long-term monitoring at 12 sites increased at 8 sites and decreased at 4 marine areas. Highest dieldrin levels were found near Richmond Harbor in San Francisco Bay, California, where DDT and dieldrin manufacturing or processing plants were located from 1945 to 1966. These data were compared to the mussel trends data, and differences were noted. Trend data could be improved with better correlation of these types of data (*59*).

Studies of the Great Lakes report the body burden of dieldrin in fish declined markedly after 1969 when these substances were banned, but concentrations since the mid-1980s have declined more slowly or plateaued (*10*). For example, studies of bloaters in Lake Michigan first detected dieldrin in 1955. Levels averaged 0.25 µg/kg until the substance was banned for agriculture in 1974. It peaked at 0.55 µg/kg in 1978 but had decreased to 0.20 µg/kg by 1986 (*60*). Surveys comparing contaminant levels in sports fish from Lake Michigan from 1985 to 1993 reported that organochlorines remained constant, with DDT having the highest concentrations, followed by chlordane and dieldrin (*61,62*). Contaminant levels in fish

Table 3. Comparative list of species: WHO estimates of potential doses of aldrin and dieldrin to the body and a qualitative description of patterns in species sensitivity.[a,b]

Taxonomic group	Aldrin	Dieldrin
Specie: aquatic organisms	96-hr LC_{30}	96-hr LC_{30}
Phyllopoda; water flea (*Daphnia magna, pulex,* and *cucullata*		-- relatively insensitive
Daphnia magna	48-hr LC_{50}	48-hr LC_{50}
Insecta: mosquito (*Aedes aegypti* and *Culex pipiens*), midge, mayfly, water boatman, dragonfly, stonefly,		(+) relatively sensitive
Pteronarcys californica (stone fly)	1.3	0.5
Malacostraca: aquatic sowbug, scud,		-- relatively insensitive
Osteichtyes: goldfish, mosquito fish, bluegill, ide, rainbow trout, medaka, flathead minnow, guppy		++ very sensitive
Salmo gairdneri (rainbow trout)	2.6	1.2
Pimephales promelas (flathead minnow)	8.2	3.8
Lepomis marochirus (bluegill)	6.2	3.1
Crangon septemspinosa (sand shrimp)	8	7
Specie: amphibians: salamander, clawed toad		(++) very sensitive
Specie: mammals (oral)		LD_{50} (mg/kg bw)
Mouse	44	38
Rat	38–67	37–87
Hamster	320	330
Guinea pig	33	49
Rabbit	50–80	45–50
Dog	65–95	65–80
Specie: avian		LD_{50} (mg/kg bw)
Dendocygna bicolor (fulvous whistling duck)	29.2 (male)	
Anas platyrhynchos (mallard duck)	520 (female)	
Gallus domesticus (domestic fowl)	25.5	
Colinus virginianus (bobwhite quail)	6.6 (female)	
Phasianus colchicus (ring-necked pheasant)	16.8 (female)	
Columbia livia (pigeon)	55	

[a]Data from Vaal et al. (*47*) and Ritter et al. (*48*).
[b]Code: -- relatively insensitive to this compound compared to other species, + relatively sensitive to this compound compared to other species, ( ) estimation based on PC model: no or few data available.

samples from the Quincy-Pasco Basin in Idaho, one of the water bodies reporting the highest dieldrin concentrations, remained the same between the 1980s and 1998 (*28,63*) (Figure 7).

Birds. Many studies have shown dieldrin to have harmful effects on birds. Birds are affected through both diet and environmental exposure. Research on herons, collected in 12 states, indicated dieldrin was the organochlorine most often responsible for death (*64*). In studies conducted in 1984 of the heronry of Charleston Harbor, South Carolina, White and Geitner (*65*) found levels of dieldrin in eggs and only a 66% survival rate of white ibises chicks. They also found high levels of dieldrin in non–fish-eating birds. Bioaccumulation in red-winged blackbirds and tree swallow eggs and nestlings correlated closely with sediment contamination around the Great Lakes and the St. Lawrence River basin (*66*).

Table 4. Trends of pollutant concentrations in Mussel Watch Project, 1986–1996.[a]

Waterbodies	Chesapeake Bay	Delaware Bay	Long Island Sound	Naragansett Bay	Tampa Bay	Galveston Bay
Dieldrin sites, no.	6	6	9	3	6	6
Sites with decreasing contaminant trends, no.	3	1	0	0	0	2

[a]Data from NOAA (*54*).

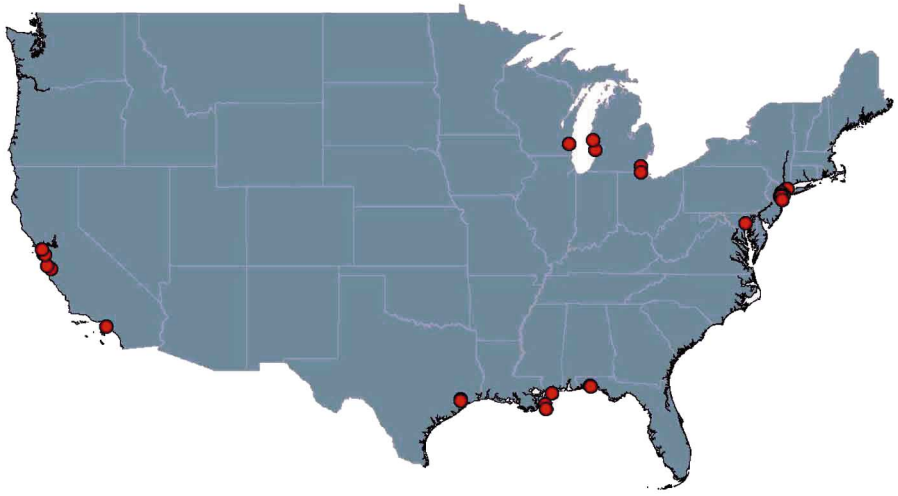

Figure 6. Locations where mussels are found with high levels of dieldrin. Map is reproduced from NOAA (*54*).

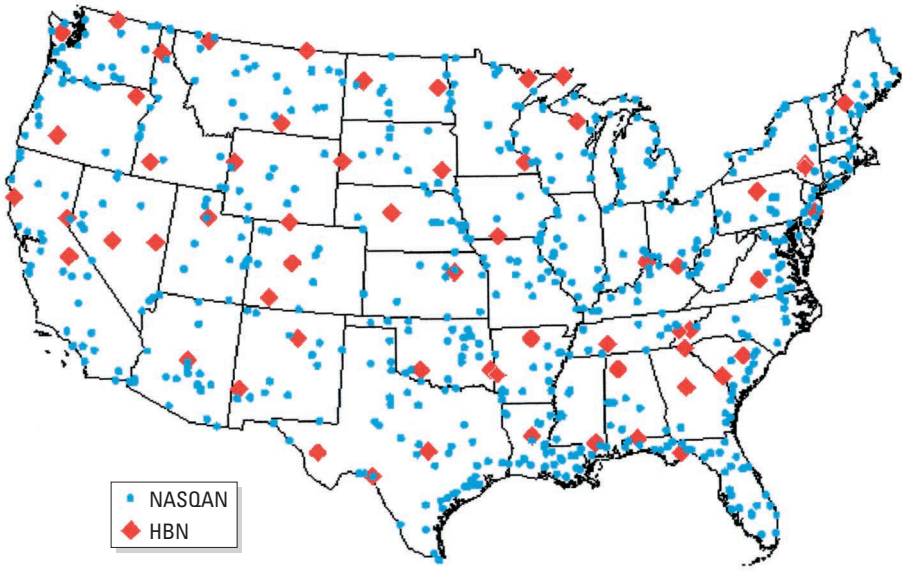

Figure 7. Historical water quality and streamflow data available from each water-quality network site indicated on the map. Map is reproduced from USGS (*28*).

Temporal trends from 1974 to 1995 in concentrations of organochlorine in 13 colonies of herring gull eggs around the Great Lakes indicated that dieldrin is decreasing as a contaminant in these bird eggs (*67,68*). Short-term changes in egg contamination in herring gulls, which deviate from long-term trends, have been associated with warm spring weather and relatively less contaminated phytoplankton in the food chain during critical periods of egg formation (*69*). Among osprey eggs collected in Maryland, Virginia, and Massachusetts in 1973 and in 1987 those at the Glen Martin National Wildlife Refuge in Maryland show a significant reduction in dieldrin contamination, but dieldrin levels in the other two states remain the same as 1970s levels. Low hatching success of osprey eggs was compared for Delaware Bay, the Maurice River in New Jersey, and the Atlantic coast. Results correlated well with the higher contaminant levels in the Delaware Bay (*70*); eggs from the Delaware Bay contained significant levels of dieldrin and other organochlorines and have reduced hatching success rates. A study of shorebirds wintering on mudflats near agricultural drains in Port Mansfield, Texas, showed 13% of the birds to be contaminated with dieldrin (*71*).

Amphibians. Amphibians exhibit high levels of biomagnification for dieldrin. Studies of common snapping turtle eggs collected from five Great Lakes basin sites from 1981 to 1991 indicate concentrations of most contaminants had decreased, but dieldrin concentrations increased during this period. Studies of 32 alligator tail muscle samples in Florida indicated the presence of dieldrin (*72*), and frog populations also had high bioaccumulation levels of dieldrin.

Marine mammals. Marine mammals are another animal group with a high BMF for dieldrin. Contamination levels are found in the lipid composition of the blubber, liver, brain, and milk fats. For example, the body weight (bw) of striped dolphins' is 17% blubber, where, research shows, 95% of the total body burden of organochlorines is found. Dieldrin is also found in liver fats. Brain fats tend to have proportionally lower levels of organochlorines than other fatty organs because the kinds of lipids found in the brain have higher amounts of phospholipid. Organs with lower levels of polar triglycerides and nonesterified fatty acids have higher levels of bioaccumulation of dieldrin.

In a study conducted from 1972 to 1991 in the Northwest Territories of Canada, blubber of ringed seals showed only minor changes in dieldrin levels, whereas DDT concentrations declined to about 20% of their 1972 levels (*73*). Marine mammals in the Northern Hemisphere tend to be more contaminated

with organochlorines than those in the Southern Hemisphere. Marine mammals in the high Arctic have higher organochlorine concentrations than those in sub-Arctic areas, possibly because of the transfer of these chemicals over the North Pole from pollution sources in Asia, Europe, and North America as well as deposition of particulate-laden Arctic haze (*74*).

Land mammals. Organochlorines were tested in water quality and mink liver assay studies conducted in Georgia and North and South Carolina from 1989 to 1991. The data collected from 141 mink found dieldrin in 64% of the animals, with a median concentration of 0.0865 µg/g. Dieldrin levels were highest among mammals in the coastal areas of North and South Carolina. This could contribute to the decline of the mink population (*75*). A study conducted from 1991 to 1995 of mink in Canada also tracked long-range atmospheric transport of dieldrin and reported concentration levels that may affect future population levels (*76*).

Evidence of distances that aldrin/dieldrin travel in the atmosphere can be seen from biomonitoring of the dieldrin-contaminated Arctic polar bears in eastern Russia, Greenland, Svalbard, Norway, and North America. Arctic areas with the highest bioaccumulation levels indicate that the source of contamination may be from North America (*77*).

Human contact: exposure. The BCF for human adipose tissue has a quantitative relationship with the lipophilicity (*n*-octanol/water partition coefficient) of dieldrin (*78*). The U.S. EPA National Human Adipose Tissue Survey (NHATS) examined 111 contaminants and collected human adipose tissues information from 14,000 people 6 months to 74 years of age across the United States from 1967 to 1992. In 1978, the survey reported it detected dieldrin in 95% of the adipose tissues samples (*79*). The survey established a baseline for average levels of these compounds. Dieldrin concentrations, like other pesticide concentrations, increased with increasing age; people 45 and older had higher levels than younger people. No significant differences were observed as related to sex or race (*80*).

The National Health and Nutrition Examination Surveys (NHANES I, NHANES II) from 1979 to 1980 analyzed blood serum samples from 5,974 people 17–74 years of age for 16 pesticides (*81*). Dieldrin was found in 17.2% of the samples. Research using these data in 1983 indicates most people are not occupationally exposed. In 1981, milk samples from 1,436 U.S. women were analyzed by gas–liquid chromatography (*82*). Dieldrin was found above the detection limit of 1.0 ppb in more than 80% of the samples collected (Figure 8, Table 5). The mean fat-adjusted residue levels for women testing above the detection limit was 164.2 ppb. The greatest percentage—88.9%—of women with dieldrin-contaminated breast milk samples lived in the Southeast. This may be caused by greater use of pesticides in the home, lawn, and garden in this area. Also, a larger proportion of homes in this area was treated to control termites.

Termite control of residences had a high statistical relationship to high body burden levels of dieldrin in human breast milk (*83*). Remediation efforts, from 1987 to 1995, of houses historically treated with dieldrin for termites indicated dieldrin levels remained the same; i.e., residences were exposed over long periods (*84*). A study to evaluate sources of exposure of small children in the home environment found dieldrin to be one of the most frequently detected pesticides. The largest number of pesticides and highest concentrations were found in carpet dust. The study found this is a danger to children because dieldrin can be absorbed through the skin (*85*).

After the National Human Adipose Tissue Survey (NHATS) ended in 1992, the U.S. EPA began to design the National Human Exposure Assessment Survey (NHEXAS) (*86*) to measure total exposure to chemicals from air, food, drinking water, and soil and dust around the home. Phase I measures only 46 chemicals for a much smaller group of 460 people living in Arizona, Maryland, Illinois, Indiana, Michigan, Minnesota, Ohio, and Wisconsin. Biologic samples are limited to blood and urine.

In 1998 the Agricultural Health Study (*87*) examined health outcomes and environmental exposures among farm families in the United States. The study evaluated food, beverage, air, dermal, dust, surface wipe, and specimen samples of blood and urine for six farm families in Iowa and North Carolina. Previous use of aldrin on the farm corresponded to

Figure 8. Sites from which dieldrin-contaminated in human breast milk samples were collected in the United States in 1981. Map is adapted from Savage et al. (*82*).

Table 5. Numbers sampled and the percentages and detection levels of fat-adjusted dieldrin found in human milk samples from nursing mothers by geographic region in the United States, 1980.[a]

Dieldrin	Northeast	Southeast	Midwest	Southwest	Northwest	Total U.S.
Number sampled	233	288	378	388	149	1,436
Percentage above detection limit	63.9%	88.9%	78.8%	85.6%	83.9%	80.8%
Levels in ppb						
0	3.4%	2.8%	7.1%	3.9%	2.0%	2.4%
(trace)	32.6%	8.3%	14.0%	10.6%	14.1%	15.0%
1–50	9.0%	4.5%	5.0%	9.8%	14.8%	7.9%
51–100	31.8%	41.0%	29.1%	32.7%	47.6%	34.7%
101–150	16.3%	16.7%	24.3%	18.3%	10.7%	18.5%
151–250	6.0%	12.8%	13.2%	12.6%	6.0%	11.1%
251–500	0.4%	9.4%	5.0%	7.7%	2.7%	5.6%
>500	0.5%	4.5%	2.5%	4.4%	2.1%	3.0%

[a]Data from Savage et al. (*82*).

dieldrin levels in the diet and in the serum of members of farm families (*87*). This finding agreed with those of a 1989 analysis of NHANES data indicating an increased risk of exposure for males that increased with age and residence on a farm (*88*).

Potential dose to the body: internal dose. The U.S. Food and Drug Administration (FDA) Selected Total Diet Study, conducted from 1991 to 1993, evaluated pesticide residues in foods prepared as they would be consumed (*89*). Of the 5,703 samples, 64% had detectable residues. Dieldrin was found in 9% of common foods. In May 2000 an update of the U.S. Department of Agriculture Pesticide Data Program reported the odds of exceeding safe daily doses of selected food–pesticide combinations. Dieldrin had the highest odds, with a 66.2% chance of exceeding safe limits from food grown in the United States (*89*).

Meat. A study in Germany (*90*) of diet and human adipose tissue, breast milk, breast cancer, and pesticide residue in blood and serum found that consumption of all meats (except fish) was the best predictor of the presence of dieldrin concentrations. Consumption of saltwater fish also correlated positively with dieldrin levels.

In the United States, sport fish consumption has been identified as a significant source of maternal and fetal dieldrin exposure (*91*). Concentrations of dieldrin are higher in fish such as croaker and surfperch, which have high lipid fats, whereas fish with low lipid levels in their muscle tissue (e.g., halibut and shark) exhibit lower contaminant levels (*92*).

The Contaminants Division of USGS in Columbia, Missouri, samples stream fish. Information is available for many streams; however, fish consumption health advisories are issued by individual states and include limited information on organochlorine contaminant levels. Fish health advisories are not regulated by the U.S. EPA or FDA (*93*). This is an important concern, as sport fish constitutes about one-fifth of the seafood eaten in the United States. It is also important to note that 25% of the world fish catch is used for fertilizer and chicken and hog feed. As fish bioconcentrate dieldrin at among the highest levels, these latter uses continue to add dieldrin contamination to the food chain.

In 1993 Environment Canada reported dieldrin was detected in 1.5% of domestic animal fats and eggs (*94*). Food sampled between 1986 and 1988 had dieldrin in avian broiler fat, in 28% of fresh poultry sausage, and in 8–15% of pork products. In the United States, dieldrin was found in 21% of pasteurized milk sampled. Dieldrin was the second most common pesticide detected in U.S. pasteurized milk in 1993.

Preparation of meats without the skin on can significantly reduce dieldrin contamination in the diet. Cooking raw fish fillets without the skin significantly reduced dieldrin content among all cooking methods. Average loss of dieldrin from prepared fish meats was 40% (*94*). A study of Canada geese in Illinois found that their breast muscle, when baked without the skin, had reduced amounts of dieldrin (*95*).

Vegetables. Using dietary exposure data from 121,700 females in the Nurses' Health Study and 51,529 males in the Health Professionals' Follow-up Study as part of an FDA Total Diet Study, investigators found that a substantial fraction of the population had dietary intakes of dieldrin in excess of health-based safety standards established by the U.S. EPA. High levels of dieldrin dietary exposure among health professionals were related to frequent consumption of summer and winter squash, 38% of the mean exposure (*96*). Other sources of dieldrin were the cucurbit family, e.g., cucumbers and melons (*51*). These vegetables absorb dieldrin through their roots into the pulp, which shows that dieldrin is present not just on the skin. A 1998 analysis determined that dieldrin contributed 46% of the toxic substances identified in Mexican cantaloupe and 94% in U.S. frozen winter squash. In 1995, an evaluation of baby food consumed by U.S. infants in their first 6 months found Federal standards for dieldrin exposure levels too high to protect infants. Dieldrin was detected at 1.0 ppb in jars of infant squash (*50,85*).

Toxicology

Accumulation transformation elimination. According to Matsumura (*97*), "dieldrin is one of the most persistent chemicals known." Dieldrin bioaccumulation is resistant to physical degradation and biologic metabolism. Like DDT, dieldrin is not easily metabolized in water and has limited capacity of being digested and excreted from the body. It is, however, easily absorbed and transported throughout the body in the blood or body hemolymph of invertebrates. In blood, dieldrin is found in the erythocytes and plasma. The percentage of dieldrin carried by erythrocytes is particularly high—39.8% compared to 12.5% for DDT. The ratio of dieldrin in a steady state in blood is rather predictable for dieldrin, fair for DDT, and poor for other chlorinated hydrocarbons. Dieldrin levels in human adipose tissue are approximately 140 times that in blood. This is because of the slower buildup of dieldrin in adipose tissue and other fatty organs that serve as storage compartments. These tissues usually have a poor supply of blood and slow establishment of extracellular water equilibrium or elimination.

As an example, areas of the body where dieldrin can accumulate were measured for dieldrin accumulation in sheep. Sheep fed 2 mg of dieldrin for 32 weeks showed a gradual accumulation of dieldrin in the liver (323 ppm), adipose tissues (126 ppm), carcass (110 ppm), heart (107 ppm), muscle (104 ppm), bones (98.6 ppm), adrenals (65.9 ppm), brain (20.5 ppm), and spinal cord (18.9 ppm) (*97*). Early studies in 1964 on mice reported high dieldrin concentrations in the gall bladder and mammary glands, and noticeable uptake in the ovaries, the central nervous system, and the cerebellum and other white matter areas of the brain. This is in contrast to DDT, which was concentrated more in brain gray matter (*98*). Concentration proportions differed among different species.

Human tissue. Dieldrin exhibits high lipid solubility and concentrates in human liver, body fat, breast milk, and semen (*99*). High levels of dieldrin have also been reported in the ovarian corpora lutea, placenta, mammary glands, and bone marrow of women (*97*). Unlike DDT and four other chlorinated hydrocarbons in which concentrations in bone marrow were lower than in adipose tissue, dieldrin has concentrations in bone marrow 19-fold higher than those in adipose tissue (*100*). The level of dieldrin in the liver tends to fluctuate as it is metabolized. Relatively high dieldrin levels in the liver fat are not as consistent as long-term low levels of accumulation in the brain.

Breast milk. Womens' adipose tissue contains dieldrin at levels approximately 30 times higher than those in breast milk. The only known way in which large amounts of dieldrin can be excreted is through lactation (*101*). In response to UNEP POPs negotiation, Greenpeace International assessed levels of dieldrin in human breast milk, standardizing a relevant testing procedure. The group also reported growing concern about contaminants crossing the placenta and affecting the fetus and of nursing infants (*102*). A study in Germany of contaminant levels in breast milk suggested that breastfeeding women should not try to lose weight until after cessation of lactation in order to reduce the amount of organochlorines fed in breast milk to their babies (*103*).

Transplacenta. Many mammal studies show that a mother's body burden is reduced as organochlorines cross the umbilical cord during pregnancy (*104*). Compared to the blood–brain barrier, which selects out contaminants, the placenta barrier is less efficient. Lipophilic molecules of dieldrin traverse by passive diffusion.

A review of human placental transfer of pesticides reported that dieldrin crosses the placenta in body fats and accumulates in the fetus, particularly in the liver, fat, and

intestines (*105*). It is also detectable in the brain of fetuses (*106*). A 1977 study (*107*) reported dieldrin concentrations in mothers' adipose tissue of 0.20 ppm; maternal blood, 0.53 ppm; placenta, 0.80 ppm; and increasingly high levels of 1.22 ppm in the fetal blood. The fetus can accumulate a body burden of dieldrin of approximately the same level as that in the mother's brain or heart. Unlike DDT, levels of dieldrin crossing over the placenta did not increase with increasing age of the mother.

Human semen. A 1989 study of organochlorines in human semen found dieldrin present in the same concentrations as that in men's blood and other biofluids (*108*).

Mammals. Contamination studies confirmed that mice and sheep also pass dieldrin across the placenta to their young. A study in newborn lambs (*109*) found dieldrin concentrations much higher in males (27.2 ppm) than in females (10.8 ppm) and the effects of contamination from suckling also higher among males (6.4 ppm) than females (5.3 ppm).

Marine mammals. The BMF for dieldrin for marine mammals is high. A large study, "Environmental Contaminants in the Marine Environment," comparing the research results of most of the current literature reported, "Dieldrin in particular is found in marine mammals throughout the world" (*97*).

The BMF for dieldrin in porpoises is second only to that for chlordane (*110*). A summary of organochlorines in marine mammals consistently reports no difference in organochlorine concentrations in immature males and females. However, as females reach reproductive maturity, dieldrin concentrations are passed to the pups. Concentrations in males increase with age. Adult males have higher concentrations of dieldrin in the blubber and liver than adult females.

The body burden of dieldrin in marine mammals seems to be inversely related to size, overall metabolic rate, and the size of lipid compartments and seems to vary with age, sex, and reproductive status. Mature males have higher levels than females. Females transfer their body burden of dieldrin over the placenta to the fetus and to nursing young during lactation. It is estimated that 26–80% of organochlorines concentrated in the blubber of females are transferred to the first-born pup. About 1–10% of the dieldrin concentrations in mothers crosses the placenta to the fetus; the remaining dieldrin is passed in lipid-rich milk. Total body burden of pups increases sharply from birth to weaning. Studies of fetal tissues indicate early differential accumulation, with organochlorine concentrations in the fetal kidney approximately 30 times higher than the relatively low concentrations in the brain and liver (*111*). Thus, dieldrin concentrations are passed from generation to generation (*112*).

Studies initiated in 1977 on pinnipeds estimate approximately 1% of the organochlorine burden occurs through transplacental transfer and 30–80% is transferred to pups through lactation (*112*). These ratios differed between dieldrin and other organochlorines and among other marine mammals. Unfortunately the research does not consistently report levels of dieldrin. Studies on harbor seals found that a much higher proportion of the female body burden for other substances can be transferred to the fetus—15% of PCBs and 30% of DDT (*113*). Baleen whales were estimated to transfer about 26% of their total body load of DDT and 14% of PCBs to the first calf, with decreasing amounts transferred to subsequent offspring.

Metastudies of marine mammals identify the difficulties in standardizing collection methods and comparing lipid fat samples within the same species or among species. They also report difficulties in relating contaminant findings to disease or reproductive failure and underscore the need for more scientific coordination (*112*). There is also a need to pool information on populations of related species in the wild, to compare the susceptibilities of different species, and to compare common datasets available on food and water contamination levels.

The Ehime University in Japan and the National Science Museum in Tokyo, both of which collect marine mammal tissues, have 1,200 specimens collected from 1976 to 1992 and now advocate formation of an International Environmental Specimen Bank (*114*). The NOAA National Marine Fisheries Service also maintains a tissue bank where historical samples have been collected and measured. For example, dieldrin measurements were taken from pilot whales stranded along the Massachusetts coast from 1986 to 1990 (*115*).

Fish. Fish retain dieldrin at higher concentration levels from food sources than from water. Dieldrin exposure at lower levels of contamination in a combination of food and water showed the highest biomagnification levels. A balance in body burden was reached more quickly at lower exposure levels than at higher levels. When dieldrin concentration was at the highest level, it was more easily excreted from the fish body. Large catfish consistently accumulated more dieldrin than small catfish but did not always retain more. Bioaccumulation in fish was found to be related to the amount of fat in muscle tissue. Fish with higher body fat retain more dieldrin. Smaller fish have higher metabolic rates, which presumably allows them to excrete dieldrin at faster rates depending on water exposure (*30*).

Dieldrin levels of fish taken from Nebraska waters varied from 0.1 to 6.7 ppm in the brain and 0.01 to 0.07 ppm in the blood. Lethal doses of dieldrin cause extremely high levels of ammonium concentrations in the brain of rainbow trout (*30*).

Birds. Concentrations of dieldrin in Japanese quail tissues and egg yolks were studied over three generations (*116*). Parents were fed a diet of 0.1–1.0 ppm dieldrin. Bioaccumulation rates in the newly hatched birds initially were higher than those of the parent for each generation, but cumulative effects were transitory between generations and remained similar in each generation. When the dieldrin diet was terminated, tissue residues declined for females, probably because of egg production, but not for males (*116*). Studies on herring gulls also confirmed that dieldrin levels of mother birds are reduced during egg production. They also found levels of dieldrin in baby birds increased from 5–15 ppm after hatching days and then leveled off (*71*). Again, scientific sampling methods should be standardized. BMFs may vary significantly by season because of weight loss and dehydration, and because measurements were made on live birds or carcasses.

Toxicologic mechanisms. The effect of organochlorines on the body is a rapidly advancing field of research. O'Shea (*112*) in his comprehensive summary of marine mammals research states the following.

> The metabolism, biotransformation, and excretion of organochlorines involves processes that convert these hydrophobic (dieldrin being one) compounds to more polar metabolites.
>
> . . . Research into the biochemical pathways of organochlorine metabolism in laboratory mammals has shown that initial steps take place on membranes of the endoplasmic reticulum of the microsomes of liver cells (hepatocytes). This is the site where the cytochrome P-450-dependent monoxygenase enzyme system function. . . . Cyotchrome-P-450 is actually a family of hemoproteins that gives a characteristic absorption spectrum of 450 nm . . . any one or more of which may be induced by a particular foreign compound (xenobiotic). Each form in turn catalyzes the oxidative metabolism of a relatively specific group of lipophilic substrates. Referred to as the mixed function oxidase (MFO) system, these biochemical pathways initially evolved to allow animals to detoxify poisonous natural compounds.

Liver. The liver is known to bioconcentrate dieldrin. Studies on the effect of dieldrin as a contaminant in the liver have been done for many species.

Fish. DDT, chlordane, and dieldrin were found to be significant risk factors in toxicopathic hepatic lesions for marine bottom-fish species. General hepatic disease increased with fish age, but sex was not a significant factor (*117*). Gilthead seabream exposed to dieldrin for 2–7 days exhibited a 100% increase in the ethacrynic acid-dependent activity of glutathione *S*-transferase (*118*). Further studies on gilthead seabream found dieldrin caused oxidative stress, highly increased activity of palmitoyl-CoA-oxidase (9.3-fold) and a nearly 2-fold increase in protein concentration of the peroxisomal fraction. Dieldrin was also found in the kidneys of fish, but tissue samples have not been collected long enough to establish trends (*119*).

Mammals. Studies of DDT and dieldrin on liver-promoting/hepatocarcinogenic actions show a strong correlation affecting gap junction-mediated intercellular communication in mice and rats liver tumors. In mice fed a diet of 10.0 mg/kg of dieldrin for 30 and 60 days, the number and volume of focal liver lesions and eosinophilic lesions increased as dieldrin was continued in the diet and subsided as dieldrin was removed (*120*).

Marine mammals. Some of the most groundbreaking research on liver metabolism has been done on cetaceans by Tanabe at Ehime University in Japan (*121*), who examined how to quantify specific isomers of PCBs and organochlorines. Tanabe's findings allowed researchers to refine tests to measure mixed-function oxidase (MFO) activity in laboratory rats, short-finned pilot whales, striped dolphins, and a killer whale to test the hypothesis that cetaceans cannot metabolize these substances. They found that cetaceans could tolerate more bioaccumulation because they have lower levels of phenobarbital induction, aldrin epoxidase, and arylhydrocarbon receptor protein, which control how highly hydrophobic chemicals like dieldrin enter cells (*122*).

Scientific groups must coordinate liver research. O'Shea, in his review of marine mammals (*112*), listed 15 studies on liver disease; only 7 of these studies reported the results of testing for organochlorine concentration levels in fatty tissues.

Tumors/Cancer. Both aldrin and dieldrin are listed on the U.S. EPA new List of Chemicals Evaluated for Carcinogenic Potential (*123*) as "possibly carcinogenic to humans." Aldrin is listed as possibly contributing to liver carcinomas, hepatic hyperplasia, and hepatocellular carcinomas. Dieldrin is suspected in the formation of hepatocarcinomas with transplantation confirmation and pulmonary metastases.

Dieldrin, aldrin, and DDT were tested to evaluate their role in inhibiting intercellular communication and metabolic cooperation between 6-thioguanine–sensitive and 6-thioguanine–resistant human teratocarcinoma cells. All three pesticides are reported to inhibit gap junction intercellular communication within the noncytotoxic range and to reduce the transfer of [^{3}H]-uridine between teratocarcinoma cells. In Chinese hamsters communication between thioguanine cells has been shown to promote tumors (*124*). Uterine leiomyoma cells *in vitro* are sensitive to ovarian hormones. An investigation of leiomyoma-derived cells from Eker rats found that dieldrin did not stimulate proliferation of uterine leiomyoma cells but did exhibit agonistic activity at transcriptional levels in the assay for estrogen-sensitive reporter genes as well as affecting progesterone receptor messages (*125*).

A 1999 review of research on cancer effects suggests that occupational exposure to aldrin, in combination with dieldrin and endrin may cause an increase in liver and biliary cancer. Aldrin may have an effect on the immune response systems as well (*126*). Caldwell et al. (*127*) found that patients exposed to dieldrin because of living near cottonfields had higher serum levels and increased incidence of childhood colorectal cancer.

Estrogen sensitivity. New scientific areas of concern beyond cancer emerged in the 1990s. These studies focused on the endocrine disruptor aspects of contaminants. Aldrin and dieldrin are listed on all lists of known endocrine-disrupting chemicals (*128*). Soto et al. (*129*) discussed this topic:

> Estrogens are defined by their ability to induce the proliferation of cells of the female genital tract. The wide chemical diversity of estrogenic compounds precludes an accurate prediction of estrogenic activity on the basis of chemical structure. Rodent bioassays are not suited for the large-scale screening of chemicals before their release into the environment because of their cost, complexity, and ethical concerns. The E-SCREEN assay was developed to assess the estrogenicity of environmental chemicals using the proliferative effect of estrogens on their target cells as an end point. This quantitative assay compares the cell number achieved by similar inocula of MCF-7 cells in the absence of estrogens (negative control) and in the presence of 17 beta-estradiol (positive control) and a range of concentrations of chemicals suspected to be estrogenic. Among the compounds tested, several 'new' estrogens were found; alkylphenols, phthalates, and some PCB congeners and hydroxylated PCBs, and the insecticides dieldrin, endosulfan, and toxaphene were estrogenic by the E-SCREEN assay. In addition, these compounds competed with estradiol for binding to the estrogen receptor and increased the levels of progesterone receptor and pS2 in MCF-7 cells, as expected from estrogen mimics.

Several studies have investigated whether a combination of potential endocrine disruptors might act cumulatively and have a synergistic effect. In 10 yeast-based assay tests for estrogen receptors, performed by four laboratories, dieldrin in a mixture with toxaphene was found to have an additive effect (*130*). A combination of these chemicals in humans and alligators inhibited binding of 17β-estradiol by 20–40%, suggesting that estrogen receptors may have more than one site for binding a synergy of chemicals (*131*). In another yeast-based estrogen receptor test, the transactivational potential of xenoestrogens and phytoestrogens was studied. Dieldrin mixed with other desethylatrazine and desisopropylatrazine chemicals showed only a weak endocrine disruptor effect. This effect possibly could be attributed to other molecular mechanisms such as metabolic modification or interference with steroidogenesis (*132*).

Reproductive difficulties. *Earthworms.* An effect of dieldrin on earthworms is structural damage to the nucleus of the sperm at low dosages, providing a test for ecotoxicity (*133*).

Water fleas. The sex ratio of *Daphnia galeata* changed when exposed to dieldrin concentrations of 300 ppb or higher. Total neonate production did not change, but the number of male fleas hatched decreased, suggesting that dieldrin affected the sex-determining system during embryogenesis. No morphologic abnormalities were observed (*134*).

Fish. Dieldrin was found in similar concentrations in the muscle of the lake trout gravid fish and in its eggs (*135*). Studies of maternal transfer of lipophilic organochlorines in salmonines and winter flounder to their eggs show a significant correlation of organochlorines in mother fish with concentrations in the eggs she lays (*63,136*). The milt of fish did not appear to be contaminated among fish with high levels of dieldrin in their fat tissues (*136*).

USGS tracks studies reporting reproductive failures among fish in the United States. Dieldrin has been found to significantly reduce populations of lake trout (*136*). There is also evidence that dieldrin is an endocrine disruptor and affects the sexual development of fish (*137*) (Figure 9).

Birds. American kestrels, fed a diet of 3 μg/g feed contaminated with dieldrin showed dieldrin contributed to significantly high concentrations in the eggs, and egg thickness was reduced by 5.0–4.8% (*138*). Studies on the presence of dieldrin in the plasma of Caspian terns indicated early reproductive failure, decreased egg viability, and increased mortality among fledglings (*139*). The presence of dieldrin is also related to breeding abnormally late in the season when the young cannot easily

find food. Also, second breeding attempts are fewer (*140*). Concentrations of dieldrin in herring gull eggs were found to vary according to egg-laying order, the third egg having the highest dieldrin concentrations.

Amphibians. Alligator studies from several lakes in Florida compared serum concentrations of 16 organochlorines, including dieldrin; sex steroid concentrations and phallus size showed some correlation. Alligators from the most contaminated lake had measurably reduced phallus size, suggesting that maternal exposure and egg viability had been significantly reduced (*141*). Among red snapping turtles, chlordane caused temperature-dependent sex reversals. When eight compounds were assayed, including dieldrin and toxaphene, significant sex reversal was reported (*142*).

Marine mammals. The transplacenta transfer of dieldrin and the passing of dieldrin from the mother's adipose tissues to the pup during lactation are documented in a number of studies among a number of species. The first pub born to a female receives the highest levels of the mother's body burden of dieldrin. The dieldrin concentration passed to pups differs significantly among different marine mammal species. Porpoises fed contaminated fish from the Walden Sea had documented trouble in reproducing.However, sea lions living in highly contaminated waters off the California coast do not appear to have a decrease in colony size (*112*).

Humans. Semen has been found to contain dieldrin in the same concentrations as male adipose tissue in marine mammals and in humans. Analysis of data from 61 studies supports significant declines in sperm density among U.S. males (the number of sperm contained in 1 mL of semen sampled). Swan et al. (*143*) reported that men tested in the United States between 1938 and 1988 showed a decrease of sperm density of about 1.5 million sperm per year. Studies conducted in Europe between 1971 and 1990 reported a sharper decrease of about 3 million sperm per year. This study concludes by recommending the "banking of semen and scrum to facilitate studies of biomarkers of exposure and trends in those exposures" (*143*).

In other studies researchers observed a significant correlation between lower sperm densities and increased levels of organochlorine substances found in seminal fluids sampled (*144*). Significant regional differences were apparent, indicating possible environmental exposure factors. In 1979, a study compared sperm concentrations among Paris males to and those in Iowa and found Iowa sperm densities to be half those reported in Paris (*145*). Iowa is one of two U.S. states where aldrin applications to the soil are the highest.

Several organochlorine insecticides have been detected in the ovarian follicular fluid of women undergoing *in vitro* fertilization (*146*). A pilot study is now under way to examine levels of endrocrine disruptors in the mother's serum, using serum collected in the 1960s, and correlating it with the sperm counts of the male offspring of those pregnancies (*147*). Studies investigating two closely related cyclodiene insecticides, chlordane and endosulfan, indicate they affect fertility mechanisms at relatively low levels (0.41 ng/mL or 0.41 ppb) (*148*). In human sperm these substances strongly inhibit neurotransmitter patterns in the amino acids active in the receptor/chlorine channels. These neurotransmitter receptor/chlorine channels are important in mammalian sperm acrosome reaction. Turner et al. (*148*) report that concentrations of cyclodinene contamination, aldrin and dieldrin listed among them, now commonly found in human and wildlife tissue are well within the range to potentially inhibit sperm acrosome reaction. Dieldrin is also known to inhibit the GABA(A) receptor/chlorine channels and glycine channels.

A report by the Royal Society of the United Kingdom in June 2000 (*149*) suggests evidence of a syndrome:

> Information available to date indicates that testicular germ cell cancers arise from precancerous, malignant gonocytes (fetal germ cells) that develop abnormally in the testis of the male fetus whilst it is in the womb . . . . It is established that disorders of development of the male in which androgen production or action are abnormal are associated with a substantial increase in risk of developing testicular germ cell cancer . . . . What the most important risk factors for testicular cancer have in common is that they are associated with disorders of androgen production or action. Both cyptorchidism and hypospadias (an abnormality of development of the penis) occur in male infants in whom androgen production or action is abnormally low. Similarly, both conditions can be induced in animals by exposing the mother during pregnancy to chemicals which can block androgen action.

Recent studies on male fetal development indicate dieldrin reduces testicular bcl-2 and changes the profile of testicular proteins *in utero.* Weeks 14–18 during pregnancy is the important period of Leydig cell hyperplasia for a male fetus and is regulated by developmental genes known to be sensitive to dieldrin. Dieldrin appears to halve human fetal testicular testosterone production (*150*). It is known that exposure to dieldrin can retard testicle descent (*124*).

Neurological mechanisms. *Rat.* Studies show dieldrin had a high affinity to stereospecific binding in the brain with GABA(A) receptors (*151*). A study investigating the effect of dieldrin on GABA(A) receptor subunit mRNA expression reported dieldrin increased β3 subunit transcripts significantly—by 300%—and decreased expression of γ2S and γ2L transcripts by 50 and 40% (*152*). Further studies on rat dorsal ganglion neurons found that dieldrin did not alter the open time distribution of the GABA(A) receptor single channels, but the mean close time was prolonged, causing a suppressive effect (*153*). This indicates that dieldrin can pose a risk to the developing brain by altering gene expression and the functional properties of GABA(A) receptors, and if these changes

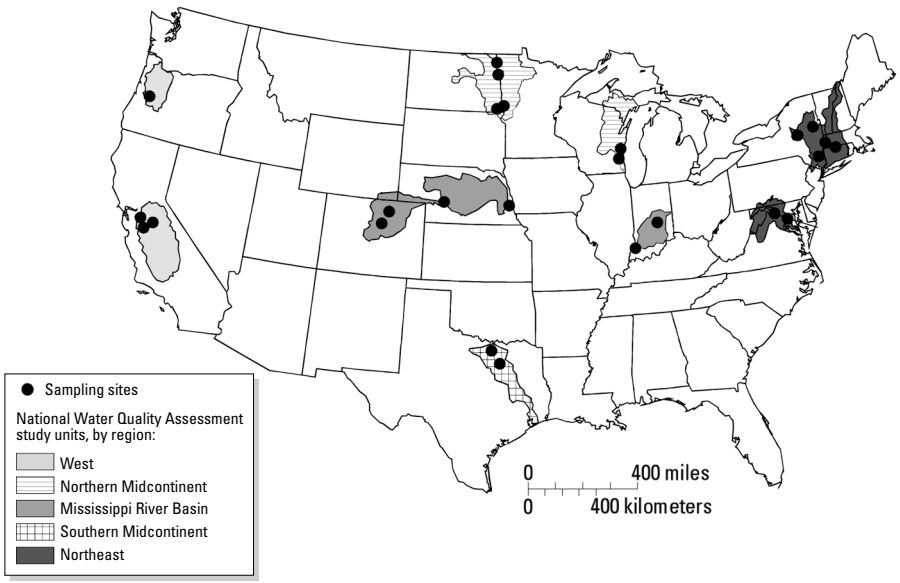

Figure 9. Areas in which potential contaminant-induced endocrine disruption has been detected in carp. Map is adapted from Goodbred et al. (*137*).

persist, there can be long-lasting effects on developing GABAergic neural circuitry and GABA-mediated behaviors (*154*). In another study of prenatal exposure of fetal rats, dieldrin and lindane altered the *tert*-butylbicyclophosphorothionate binding to GABA(A) receptors in the brainstem, with possible behavioral consequences (*155*). Albrect (*156*) found dieldrin to have the potential for causing an increase in general central nervous system excitability in mice by acting as a neurotoxin potentially inhibiting pentylenetrazol signaling that may cause or relate to persistent behavioral stimulation (*156*). Behavioral tests on rats treated with aldrin *in utero* showed the time for incisor teeth eruption was delayed and the time for testes descent increased. When rat pups became adults, they showed higher locomotor frequency measured by total number and duration of head-dips even when pesticide was not detected in the animal's tissue (*157*). Primary neuronal cultures of mesencephalic neurons from fetal rats and mice exposed to 7.98 µM of dieldrin showed a decrease in dopaminergic neurons and potential neurodegeneration (*158*).

Amphibians. Exposure of toad embryos to dieldrin induced hyperactivity in the swimming larvae (*159*). Frogs, *Xenopus laevis,* exposed to low concentrations of dieldrin for 10 days while in the embryo–larval stage had spinal deformities (*126*).

Marine mammals. The Environmental Conservation Division of NOAA reported low concentrations of organochlorines, including dieldrin, in brain tissues of stranded pilot whales, relative to levels in other tissue levels of bioconcentrations This may be related to the disparate lipid composition in this tissue as well as the presence of a blood–brain barrier. However, the levels of dieldrin reported in marine mammals' brains suggest possible problems in neurologic development (*115*).

Genetic susceptibility. Little scientific research has been published on genetic mutations and dieldrin.

Table 6. Typical levels of dieldrin in breast-fed infants, 1980.[a]

Substance	Typical levels	FDA action levels for cows' milk	Allowable daily intake	Daily intake of parent-fed infants
Dieldrin	1–6 ppb	7.3 ppb	0.1 µg/kg	0.8 µg/kg

[a]Data from Rogan et al. (*160*).

Biologically effective dose. In 1980, Rogan et al. (*160*) published a table of typical levels of dieldrin in breast-fed infants. This article also discussed difficulties encountered in estimating fetus and infant exposures to dieldrin because some women's milk is more fat-rich than others. The research had little data on whether the mothers were nursing their first babies or had other children, which could have reduced their body burdens. However, estimates made in 1994 indicated that children whose mothers had high exposure rates to dieldrin, when breast-fed for 9 months, had increasing risks of dieldrin exposure and potential cancers. Comparing exposure levels of dieldrin in human milk samples reported in Table 5 with those in Tables 6 and 7 reveals that a high percentage of women—63%—had dieldrin concentrations in breast milk over FDA action limits for cows' milk.

Epidemiology

More work remains to be done to coordinate environmental information and epidemiologic studies relating population exposure with specific diseases. Below are some of the diseases for which there is evidence that dieldrin may be one of the causal or contributing factors.

Early expression of disease and health effects. **Reproductive health.** Reproductive health failures have been reported in fish, birds, alligators, harbor seals, mink, and humans. A study by Chandra and Stephen (*161*) from 1982 to 1995 reports that there has been a substantial increase (21.4%) in impaired fertility among U.S. women. The greatest increase (41.9%) is reported among women under 25 years of age. The presence of dieldrin in the human fetus, eggs, and semen is now being actively studied. In 1981, Dougherty (*144*) investigated the relationship between the Atlas of Cancer Mortality and toxic exposure, indicating strong evidence of environmental factors. They measured organochlorines in male sperm as a good burden indicator. The study reports organochlorines significantly reduced male sperm density, suggesting that these substances decrease the cell division rates by causing DNA damage and potentially lead to cell mutation and cancer.

Human breast cancer. Breast cancer incidence increased from 1970 to 1990 in the United States by 40% for women 60 years of age or older, and by 5% for women younger than 50 (*162*). Breast cancer affected one in eight women in the United States in 1999 (*163*).

Hoyer et al. (*164*) published a study in 1998 of 7,712 Danish women observed over 17 years. The study indicated that women with concentrations of dieldrin in their blood had a 200% increased chance of having breast cancer (*164*). A follow-up study published in May 2000 (*165*) indicated that dieldrin also affects women's breast cancer survival rate. The authors found that women with higher serum dieldrin concentration levels who contracted breast cancer also had a poorer survival rate and did not respond as well to standard treatment methods, suggesting the exposure to organochlorines somehow induced the aggressiveness of the tumor. A study in Cape Cod, Massachusetts, workers exposed to dieldrin through their occupations showed no increases in breast cancer (*166*). However, the maps delineating areas with breast cancer mortalities and those showing areas with high dieldrin exposure have a high degree of correlation (*167*) (Figure 10).

In 1994, Bernstein et al. (*168*), conducting research on the National Health Nurses' Study, found physical exercise, which alters the production of ovarian hormones and lowers body weight appeared to be related to a reduced rate of breast cancer (*168*). It is not known if these women also had lower levels of organochlorines in their tissues. It is known, however, that lower levels of body fat have lower bioaccumulation of organochlorines in the blood serum. This was demonstrated 5 years earlier, in 1989, when studies among marine mammals reported that animals with elevated rates of metabolism and lower levels of fatty tissue had lower bioaccumulation of organochlorines (*169*). It is not known how this affects the onset of breast cancer. However, this is an example of how research across disciplines could complement each other.

Colon/rectal cancer. Colon and rectal cancer is the third most common type of cancer in the United States for both males and females, affecting 47.1 people in 100,000. Incidence rates increased until 1973 and may now have peaked for both white males and female. There has been a gradual decline in the incidence of colon and rectal cancer among black women, but the incidence

Table 7. Estimated excess risk of cancer by concentrations of chemicals and duration of breast feeding, 1994.[a]

	Concentration of chemical in breast milk, ppm (fat)		Total dose, mg 9 months		Average daily dose, mg/kg/day	Excess risk ($\times 10^{-5}$) by duration and level of infant exposure			
Substance	50th percentile	90th percentile	90th percentile	mg/kg/day^{-1}	9 months 90th percentile	6 weeks median	3 months 90th percentile	6 months median	9 months 90th percentile
Dieldrin	< 0.01	0.12	0.8	20.0	3.6×10^{-6}	0.02	3.48	0.03	7.15

[a]Data from Scheele (*100*).

among black men continues to increase. From 1973 to 1991, colon cancer rates increased by 0.3% for all people (*167*) (Figure 11). Agricultural use of aldrin was the highest in Iowa. Interestingly, Iowa also has some of the highest national rates for colon cancers. At the same time it has among the lowest rates of testicular cancer, and it is an area where low sperm densities have been reported. Similarly, in a study of 570 workers at a pesticide plant from 1954 to 1970, there was no increase in liver cancer, but there was an increase of number of deaths from rectal cancer that was inversely related to the dose gradient (*170*) (Figure 11). More research could be done on the potential disease outcomes of high or lower dieldrin exposure levels.

Testicular cancer. The incidence of testicular cancer has risen in the United States 42% from 1973 to 1991. Highest rates are reported in Seattle, Washington; Hawaii; and San Francisco–Oakland, California (*167*). Testicular cancer is the most common form of cancer among young men in the United States. Testicular cancer incidence in Ontario, Canada, part of the Great Lakes area, increased 59% between 1964 and 1996, for seminoma and non-seminoma cancers (*171*). There appears to be a correlation between low sperm counts, hypospadias, and testicular cancer.

Atlanta, Georgia, has among the highest levels in the United States of penis malformations among male infants (*172*). The city has also been identified as one of the areas with the highest dieldrin exposure levels in the country. USGS (*20*) reported that during 1994–1995, dieldrin was found in 5 of 37 shallow groundwater wells. This may be a result of termite treatment of houses in the vicinity and use of dieldrin in the textile industry. The source of the city's drinking water is surface water from the river. The possible relationship of hypospadias with dieldrin, which is an endocrine disruptor substance, is of increasing scientific interest (*104,173*).

Parkinson's disease. Parkinson's disease affects more than one million people in the United States. Each year an additional 60,000 people are diagnosed with the disease. Parkinson's affects the elderly, but there is an increasing trend for the disease to affect a younger population—approximately 15–20% of those diagnosed are under 50 years of age; 10% are younger then 40 (*174,175*).

A 10-year study conducted at UCLA tracking 22,700 people with Parkinson's, disease between 1984 and 1993 (*176*) found that people living in rural areas who were identified as having the highest rates of pesticide use, died of the disease approximately 1.5 times more often than residents of urban communities. Seventy percent of these people with Parkinson's disease had lived in the same rural area with high pesticide use for more than 20 years (*176*).

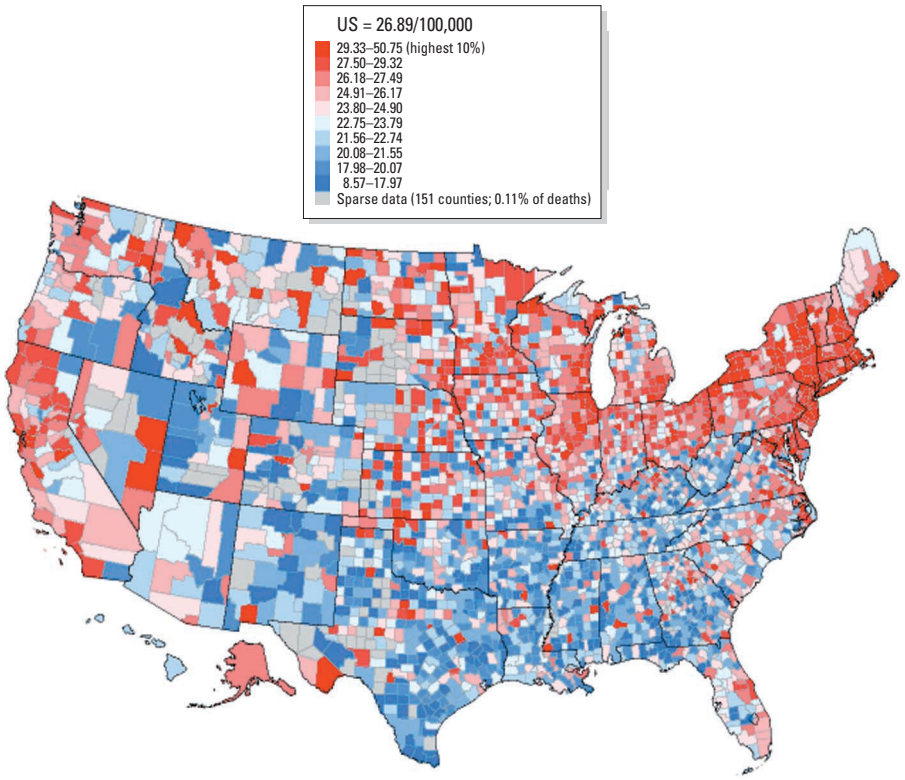

Figure 10. Breast cancer mortality rates, white females, 1970–1994. Map is reproduced from SEER–National Cancer Institute (*167*).

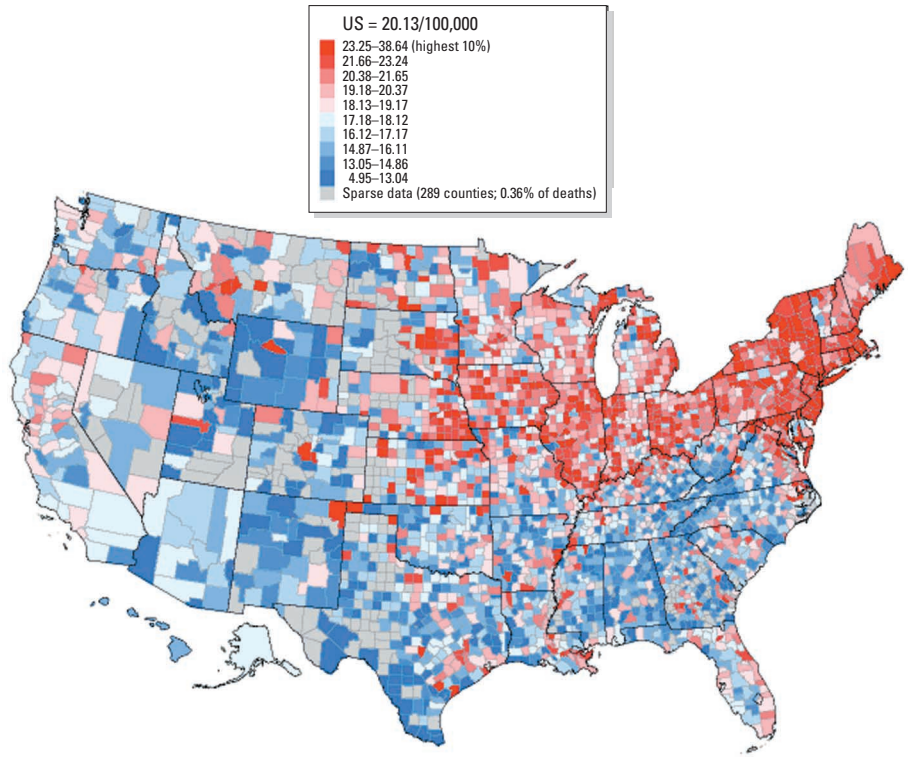

Figure 11. Colon cancer mortality rates, white males, 1970–1994. Map is reproduced from SEER–National Cancer Institute (*167*).

Table 8. Government databases tracking environmental deposition and effects of aldrin and dieldrin.

Database location	Aldrin–dieldrin profile
Producers: incomplete database U.S. EPA *http://www.cdpr.ca.gov/cgi-bin.epa/comp.pl?pccode* Industrial discharge info *http://environet.policy.net/health/neighborhood/cehfuture/*	65 companies report production in United States. 35 tracked by U.S. EPA website.
Volume: no data; report no longer produced U.S. Internationall Trade Commission: *Synthetic Organic Chemicals* (*13*); produced from 1918 to 1996	Estimated volume of aldrin/dieldrin. In the 1960s, aldrin/dieldrin were the second largest agrochemical used in United States.
Trade: large trade baskets Canada: *http://strategis.ic.gc.ca/cgi-bin/tdst-bi*	Exports of chlorinated hydrocarbons are reported as 3.9 million pounds in 1994 and 1.7–3.9 million pounds in 1996.
Soil monitoring: Poor access to national data U.S. EPA: maps in draft report only *http://www.epa.gov/glnpo/bnsdoc/* USGS: no data on aldrin/dieldrin *http://ca.water.usgs.gov/pnsp/use92/* California: Good California data by county *http://www.cdpr.ca.gov/doc/pur/.htm*	Aldrin use was concentrated in the Midwest. Iowa and Illinois alone accounted for 59% of the total acreage treated in 1971. Aldrin was applied to Iowa soils at the rate of 5–6.5 million pounds between 1961 and 1965. States outside the Midwest accounted for only 2.7% of the aldrin applied to corn in 1966, declining to 0.5% in 1971. Dieldrin distribution patterns differ. The Northeast, in 1966, used proportionally more dieldrin than the rest of the country. The Northeast used only 0.2% of national aldrin sales but 6.1% of national dieldrin sales on crops. Large amounts of dieldrin were used in cherry orchards in Indiana. Urban and mixed-use areas in the South also showed high use of dieldrin for termite control, and home and lawn products.
Cold deposition: good temperature data maps World water and climate atlas *http://atlas.usu.edu/download.htm* Climate change research section *http://www.cgd.ucar.edu:80/ccr/*	Aldrin and dieldrin volatize and "grasshopper" into cooler climate areas where they collect and are discharged in the spring thaw.
Air monitoring: Good mapped with data set per site U.S.EPA *http://www.epa.gov/grtlakes/iadn/results/1997/1997_DIELD_TBLE.html*	Dieldrin detected at all monitoring stations. Trends indicate it will disappear from the environment by 2030.
National Priority List sites: only at state level U.S. EPA *http://www.epa.gov/glnpo/bnsdoc/*	Aldrin is detected at 69 NPL sites, dieldrin at 101 sites. Aldrin is reported at 20 hazardous waste landfills, dieldrin at 29 nationwide.
Stream bed sediment USGSC: composite OC; no aldrin/dieldrin maps *http://www.water.usgs.gov/lookup/get?nawqapest* *http://water.wr.usgs.gov/pnsp/rep/fs97039/sw4.html*	In1993, studies show dieldrin was more frequently detected than the other 26 insecticides studied in 1,016 sites located at 50 of the major river basins of the United States. Sampling from 50 large U.S. river basin networks indicated that 90% of water and fish samples contain organochlorines. DDT and dieldrin are the most prevalent.
Oceanic plankton: no data NOAA *http://www.chbr.noaa.gov:80/CoastalResearch/Map2A.htm*	Dieldrin in vegetation research is poor.
Mussels: mapped by monitoring site NOAA: National Status and Trends Program *http://ccmaserver.nos.noaa.gov*	Dieldrin found in mussels at 45 sites; high levels of contamination are reported at 22 sites in 1993.
Fish: scattered information not mapped NOAA: National Benthic Surveillance Project (NBSP) *http://www.nwfsc.noaa.gov/pubs/93pub/93toc.html* USGS: *http://www.water.usgs.gov/lookup/get?nawqapest* *http://biology.usgs.gov/geotech/documents/ecrc.html* U.S. EPA: AQUIRE *http://www.epa.gov/med/databases/aquire.html*	Dieldrin BMF for fish very high. Dieldrin is detected in fish at 50% of the sites sampled by USGS, at 30% of the sites exceeding human health guidelines, and at 19% of the sites exceeding the limits of New York guidelines for protection offish-eating wildlife. Dietary studies confirm that saltwater and freshwater fish are highly contaminated. Marine and freshwater fish and sediment studies have highly correlated contamination levels
Marine mammals: not mapped USGS: book—Biology of Marine Mammals, 1999 (*112*) NOAA: Environmental Conservation Division Japan: International Environmental Specimen Bank, National Science Museum, Tokyo	Dieldrin is one of the most common contaminant in marine mammals around the world. It is passes up the food chain and transferred to the fetus and the next generation; 26–80% of organochlorine concentrates in female blubber transfer to infants; 1–10% passes transplacenta to the fetus; the rest in breast milk.
Birds: No central database USGS: Colombia, Missouri *http://www.ecre.usgs.gov/data/ncbp/ncbp.html* USGS: Patuxant, Maryland *http://www.pwrc.usgs.gov/*	Dieldrin found in a large range of bird species and eggs.
Amphibians North American Amphibian Monitoring Program *http://www.im.nbs.gov/amphibs.html*	Amphibians show a high level of biomagnification for dieldrin. Common snapping turtle eggs in five Great Lakes basin sites collected from 1981 to 1991 indicate concentrations of most contaminants had decreased, but dieldrin concentrations increased during this time. Studies of 32 alligator tail muscle samples in Florida also indicated concentrations of dieldrin. Frog populations also showed high bioconcentration levels for dieldrin and had spinal deformities.

(Continued)

Table 8. Government databases tracking environmental deposition and effects of aldrin and dieldrin.

Database location	Aldrin–dieldrin profile
Vertebrates in estuaries: good data sets USGS: Patuxant, Maryland *http://www.pwrc.usgs.gov/bioeco/mink.htm*	Dieldrin found in the adipose tissue of mink along the Eastern seaboard, Iowa, Minnesota, Ontario, and British Columbia.
Human exposure: only available in reports Adipose tissue: U.S. EPA National Adipose Tissue Survey (*86*) Breast milk: U.S. EPA Government Accounting Office evaluation *http://www.gao.gov/daybook/00503.htm*	Dieldrin found in the adipose tissue of 95% of the 28,000 people sampled in 1978 and in the breast milk of more than 80% of 1,435 women in 1981.
Human exposure: good data sets, but for fish. Dietary risk: National Health and Nutrition Evaluation Survey (*86*) U.S. Department of Agriculture *http://www.consunion.org* (go to "Do You Know What You are Eating?") *http://www.ecologic-ipm.com/PDP/Table91998.pdf*	Dieldrin found in the diet of 17.2% of 5,994 people surveyed. Dieldrin found in 80% of summer squash, and in cucumbers, melons, and soybeans. The chance of eating dieldrin in common foods is 66%. Dieldrin was the second most common pesticide detected in U.S. pasteurized milk in 1993.
Toxicology: endrocrine disrupter Fish USGS *http://water.wr.usgs.gov/pnsp/rep/carp2/fl.html* Humans DataCHEST?ChemADVISOR *http://www.datachest.com* Tulane *http://www.tmc.tulane.edu/cbr* UK/Royal Society *http://www.royalsoc.ac.uk/templates/statements/StatementDetails.cfm?statementid=111* Japan *http://www.eic.or.jp/eanet/e/end/sp98.html*	Listed as a potential endocrine disrupter.
Toxicology: neurologic no central database	Dieldrin found in spinal cord and brain tissues.
Epidemiology: No database Reproductive failure: No database Breast cancer: *http://www-DCEG.IMS.NCI.NIH.GOV/cgi-bin/atlas/mapview?direct=brecwf50* *http://cfe.cornell.edu/bcerf/* *http://www.bestiary.com/ibc/index.html* Liver cancer: *http://www-DCEG.IMS.NCI.NIH.GOV/cgi-bin/atlas/site-discuss?site=tes* Testicular cancer: h*ttp://www-DCEG.IMS.NCI.NIH.GOV/cgi-bin/atlas/mapview?direct=tesswm70* Colon cancer *http://www-DCEG.IMS.NCI.NIH.GOV/cgi-bin/atlas/mapview?direct+colcwm70* Parkinson's disease: California Rural Health Department of Epidemiology, School of Public Health, UCLA. e-mail: britz@ucla.edu	Dieldrin is a potential biomarker for diseases.

Small sample studies have shown residues of dieldrin present in the brains of 6 of 20 Parkinson's patients (*177*). Dieldrin is reported to deplete brain monoamines and promote dopaminergic neurodegeration similar to what happens in Parkinson's disease (*178*). Another study reported acute cytotoxicity of dieldrin in PC-12 cells and an association with the onset of Parkinson's disease (*179*).

Conclusions

Aldrin and dieldrin, two substances whose U.S. registrations were cancelled and products withdrawn from the market more than 20 years ago, are still the focus of a great variety of scientific research. This illustrates and expands the concept that these chemicals are persistent. Persistence is defined in agroeconomic terms as the half-life of a substance in the soil. For aldrin and dieldrin this has been determined to be 2–15 years. Environmental scientists engaged in air particle monitoring and in measuring plant and animal bioaccumulation now estimate that dieldrin, introduced in 1950, will be present in the environment until 2030, almost a century after its introduction.

This article confirms that Lioy's graph (*21*) (Figure 1) is a useful way to review the scientific literature on aldrin and dieldrin. It provides a way to examine some of the data gaps, some of the parallel scientific findings, and evidence of where more collaboration between scientist groups might improve and accelerate research in this field.

Data Coordination

A major gap in data used to evaluate chemicals in the United States is the lack of any U.S. government database reporting amounts or volumes of chemical substances being manufactured each year. In 1994, the U.S. Congress dismantled the reporting mechanism of the International Trade Commission (*180*). The Commission collected and published annual data on the total volume of production for each chemical substance manufactured in the United States; it was the central source of government reporting. The International Trade Commission production data began to be reported in 1917 in *Synthetic Organic Chemicals* (*13*) and and was published annually for the next 78 years. With cessation of publishing these data in 1994, there now is no central data source on how much aldrin and dieldrin are produced annually in the United States. Congress apparently believed that private reporting companies would replace this U.S. government function. Among the three private sources reviewed in this article (*8,24–26*) (Table 2) there are substantial

differences and data gaps, including the database U.S. EPA maintains on companies identified as producers. None of these data sources publish data on the volume of substances being produced or sold. Companies are not required to report to the U.S. government the quantities of substances exported if they are not shipped as pure substances, which is only about 25% of the agricultural chemical substances shipped each year.

Data collection by separate government organizations could be greatly improved with greater coordination and mapping. USGS has done a commendable job combining contamination data for stream sediments, rivers, and groundwater. They have also produced a compendium of studies on contamination among marine mammals (*112*). These studies would be more useful for other researchers if the data could be mapped. The NOAA's Mussel Watch Project (*54*), which does provide latitude and longitude data maps, could be combined with marine fish and sediment data and USGS freshwater stream sediment contamination data (*28*). USGS has just begun to isolate specific stream reach data and to provide information on the Internet about contaminants like dieldrin (*28*). The U.S. EPA now does an commendable job of providing on the Internet access to air particle data for dieldrin, by geographic area, from each of their monitoring stations on a seasonal basis (*43*). USGS is now working on producing fish contaminant data in map format (*181*). USGS freshwater fish data, U.S. EPA aquatic data on the AQUIRE and ECOTOX databases and NOAA marine toxicity data could be substantially enhanced with more coordination among agencies (Table 8).

Policy Coordination

Serious public policy problems can occur when one agency defines its task too narrowly and adequate provisions are not made to transfer responsibility to another authority. When funding was cut for U.S. EPA studies on human adipose tissue and breast milk during the 1980s, no continuation strategy was in place for the Department of Health and Human Services to assume responsibility for these studies once U.S. EPA made the determination to eliminate them. This left large test populations, with more then 80% having dieldrin concentrations in their adipose tissues and breast milk, with no follow-up program to monitor long-term heath effects. This situation is now the subject of a government General Accounting Office report (*86*) published in May 2000. The report states:

> Of particular concern at the federal level, is that coordinated, long-term planning among federal agencies has been lacking, partly because of sporadic agency commitments to human exposure measurement and monitoring. HHS [Health and Human Services] and EPA officials indicated that they have been discussing the merits of establishing a coordinated interagency human exposure program, but they have not yet formalized or agreed upon a long-term strategy. A long-term coordinated strategy should also ensure adequate linkages between collection efforts and agencies'goals, provide a framework for coordinated data collection efforts that consider agencies' needs and expertise, provide a framework for identifying at-risk populations, and consider states' needs for information. (*86*)

Another public policy issue concerns providing food security. The recent study of dieldrin food contamination conducted using Department of Agriculture data recommends farmers be made more aware of the health dangers of growing squash, melon, and soybean crops in dieldrin-saturated soils (*52*). Public health advisories must be more accurate and report dietary concerns for specific areas and for sport fish. Correlation with USGS stream contaminant data could also be useful to U.S. EPA for monitoring and protecting drinking water intake systems at risk.

It is unfortunate that aldrin and dieldrin, once used in very high volumes in the United States cannot be tracked by USGS agriculture chemical usage maps (*29*). Mapping dieldrin-saturated areas would greatly improve access to useful information to improving food security. Database coordination could be achieved by combining information on chemicals used for agricultural field applications and termite control with USGS stream data. These data are now available only on a state basis, collected in a U.S. EPA report (*10*).

These kinds of data must be more systematically incorporated into public health studies. For example, in Minnesota, a state for which environmental databases report high levels of dieldrin in almost every category, an extensive children's dietary study is now under way; however, dieldrin is not one of the contaminants included in the research design. Making environmental deposition and fate information more accessible will greatly enhance the conduct of more effective human health surveys as well as to target areas where more extensive studies of human adipose tissue and breast milk are needed.

Scientific Collaboration

Aldrin and dieldrin clearly have effects on the food chain. Mechanisms of effect would be better understood if there were more central data collection reporting systems. Data on the bioaccumulation levels specific for species and different BMFs among species need to be made more accessible. WHO reports some of these data. Both the U.S. EPA and USGS have in the past year actively begun to renovate their databases to improve user access.

More scientific collaboration is needed to establish protocols on how to sample and report data on bioaccumulation and evidence of disease. Bioaccumulation levels, if not systematically collected, cannot be used to correctly assess contamination levels within species and trends in disease or reproductive health. For example, fatty tissue sampling should be reported with regard to the age, sexual maturity of females, seasonal changes in diet and and the amount of fat of the animal. Breast milk samples must be evaluated in light of whether this is the mother's first or subsequent offspring as well as differences in mother's body burdens, fatty tissues, and metabolism rates. Investigators conducting research on liver abnormalities in humans and in marine mammals need to more consistently report organochlorine contaminant levels or the research findings are limited for further use. Not enough research has been carried out or tissue samples collected for evaluation of semen and testicle tissue contamination levels and their relationships to dropping fertility rates or increasing rates of testicular cancer.

There appear to be biologic gaps in the research on dieldrin contamination. For example, there is little research on dieldrin contamination levels in plant mechanisms. This is particularly evident for plankton and its probable contributing role of biomagnification in the food chain. NOAA's Plankton Disease Monitoring System now being set up should expand its mission and usefulness to other scientists by including monitoring of organochlorine concentrations (*182*).

Table 3 identifies some of the major scientific research groups working with aldrin and dieldrin. A review of scientific databases indicates the United States has a high capability to report the environmental deposition and fate, exposure, and toxicology of organochlorines. Many of the research groups listed are now actively improving their databases and providing better access to them on the Internet. The U.S. government over the last 20 years has built up a range of long-term databases within the structure and function of each agency's own legislated mandated. The challenge is for Federal agencies with separate scientific research groups to reach out to other disciplines and to make their databases more interactive.

The relationship of environmental exposure and public health is now becoming a very active field of research (*183*). It is apparent from the data sources reported in Table 8 that dieldrin remains in the environment at high levels, and dieldrin exposure, therefore, raises significant issues for public health. Tools to find and use information are rapidly improving. Dieldrin has been used in this review as a "tracking device" for other researchers to investigate environmental contamination.

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